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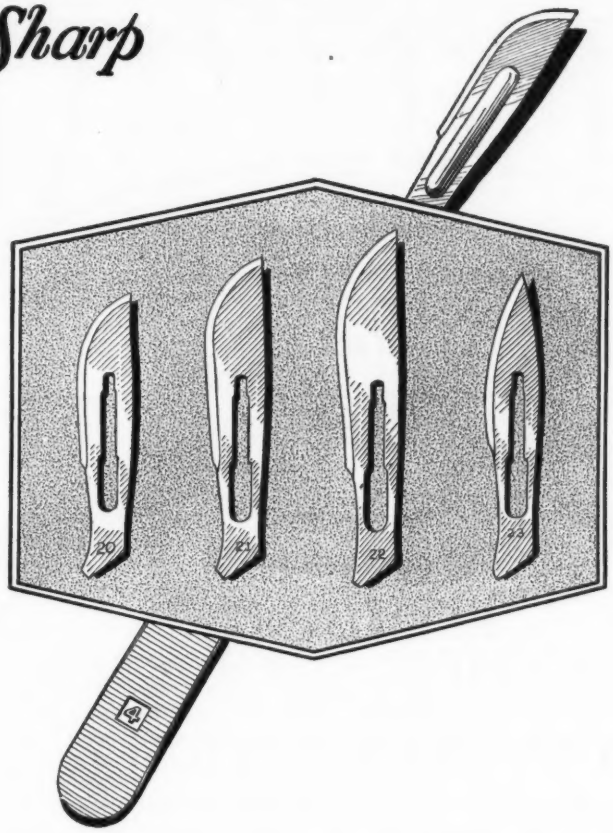

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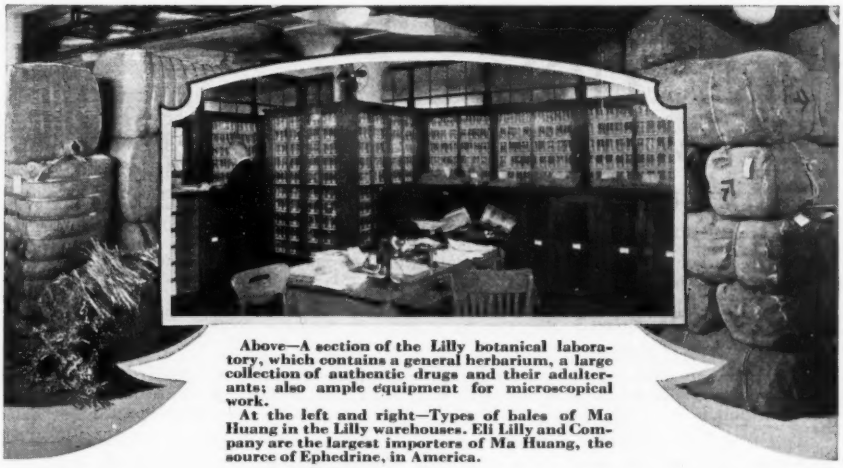

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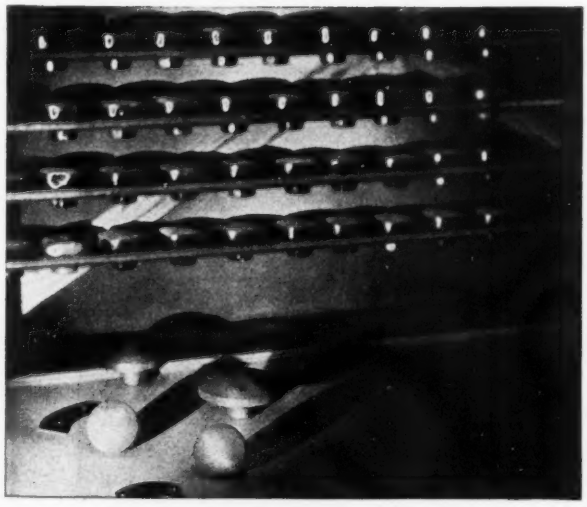

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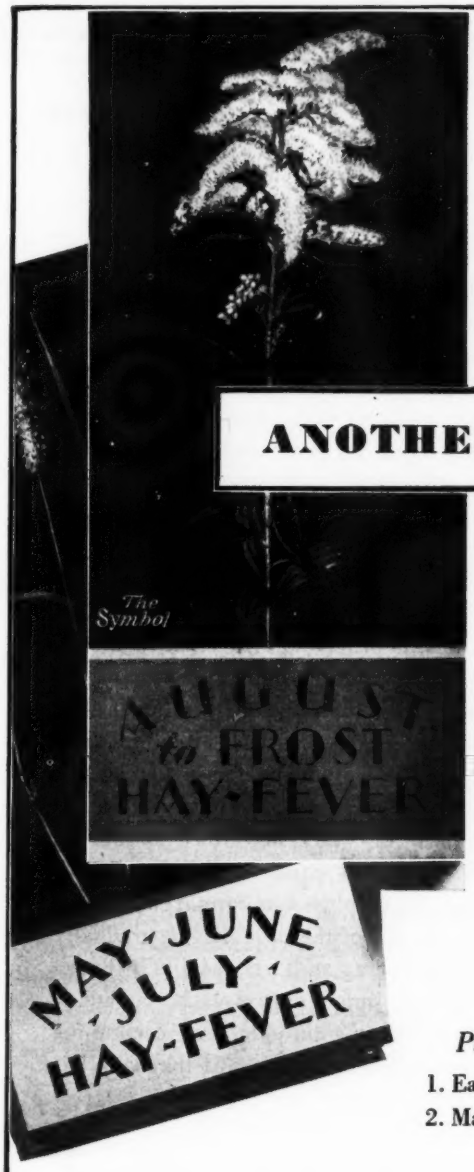

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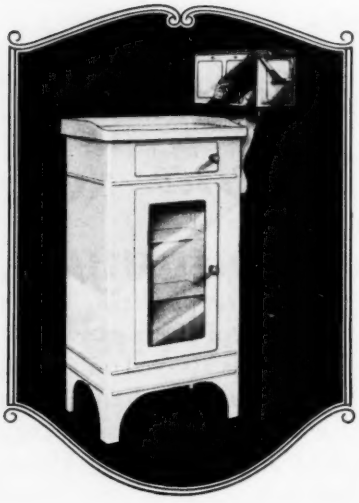

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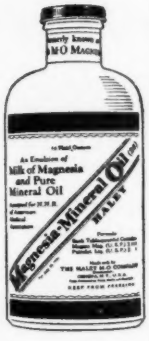

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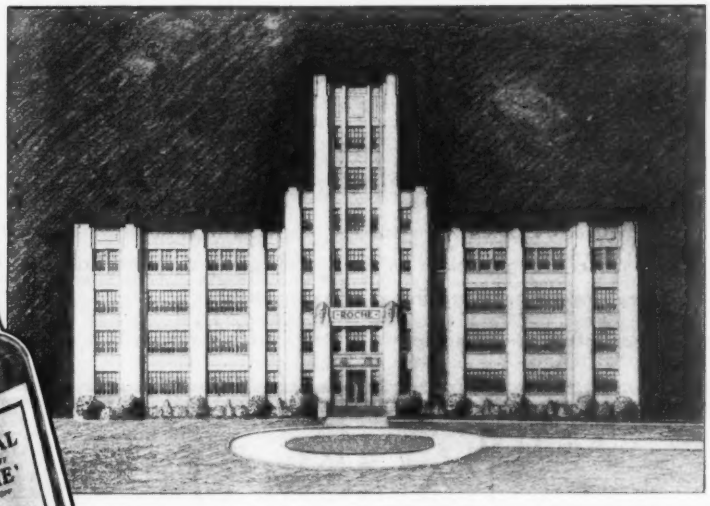

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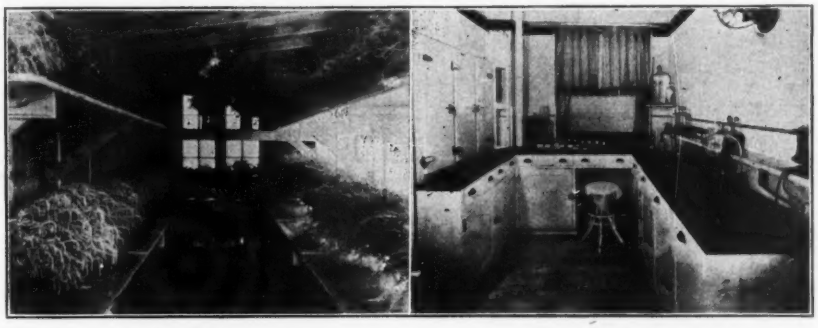

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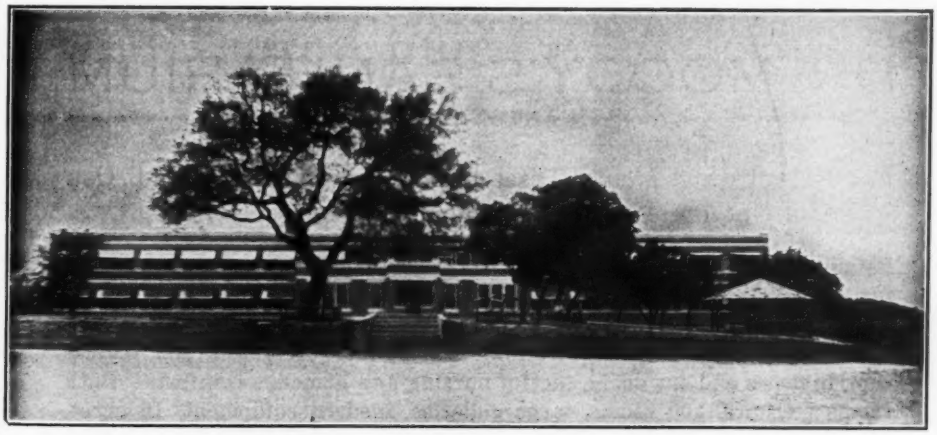

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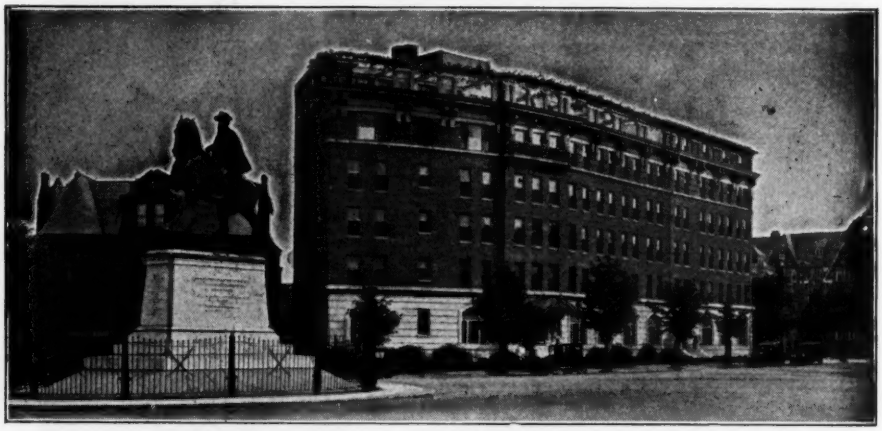

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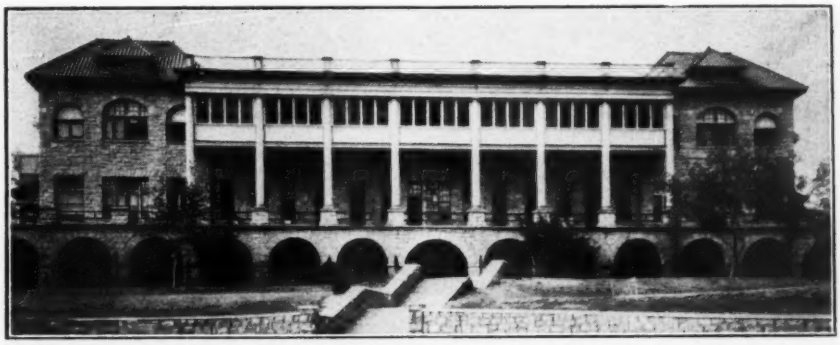

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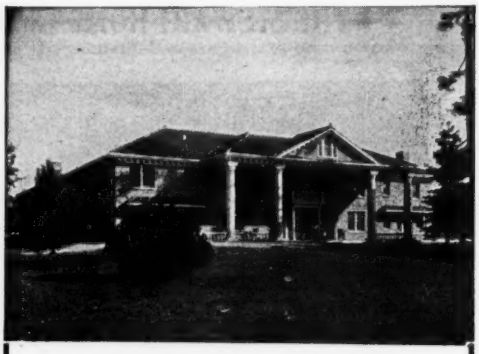

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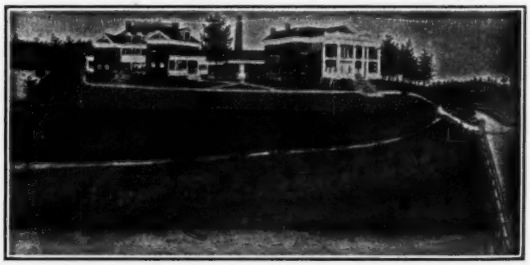

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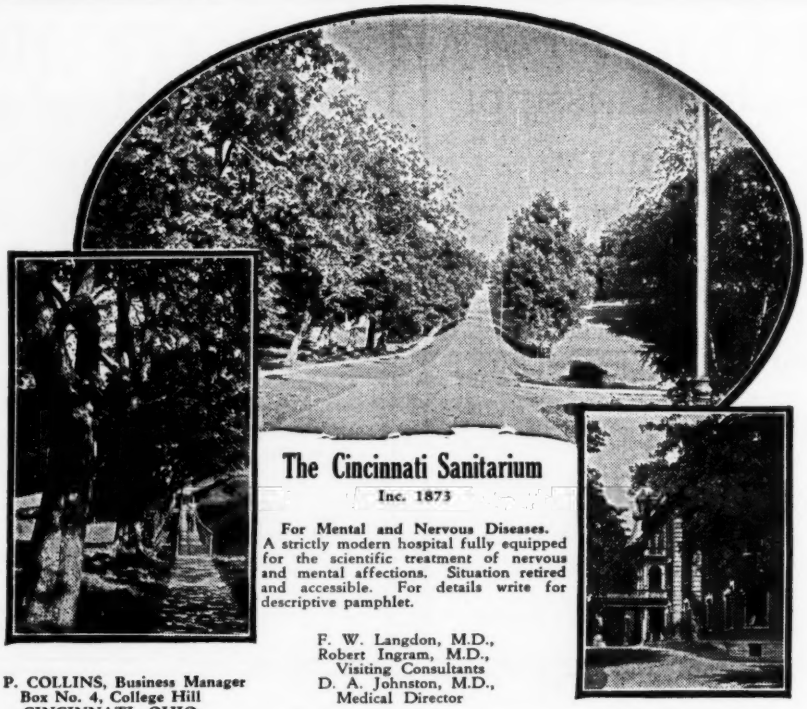

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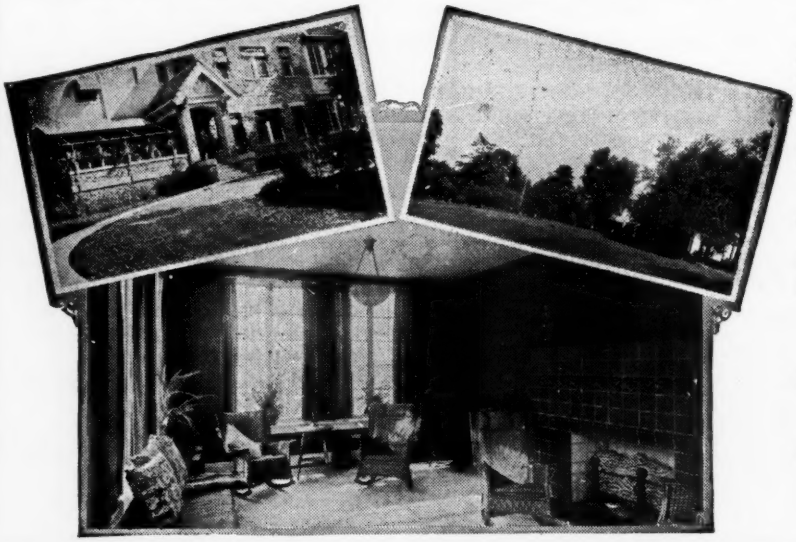

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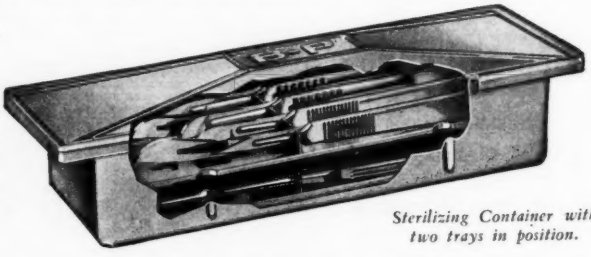

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POISONOUS SNAKES AND THE ANTI-VENIN TREATMENT*

By COL. M. L. CRIMMINS,
Fort Sam Houston, Texas

We have in the United States eleven species of rattlesnakes belonging to the *crotalus* family, two species of pigmy rattlesnakes belonging to the *sistrurus* family, and two species of moccasin of the *agkistrodon* family, the cotton-mouth moccasin and the copperhead or upland moccasin. They all belong to the pit-viper family and can be distinguished from non-poisonous snakes by the following signs:

(1) They have triangular heads, thin necks, and stout bodies.

(2) A blind pit on each side of the nose between the eyes and nostrils.

(3) Elliptical pupils like a cat.

(4) Large fangs that fold back against the roof of the mouth, in white membranous tissue sacks.

(5) Single scales on the under surface. Harmless snakes have double scales from the vent to the end of the tail.

Coral snakes are also poisonous and belong to the *micrurus* family. They are the Harlequin (*micrurus fulvius*) of the South and the Sonora coral snake (*micrurus euryxanthus*) of the Sonoran region. They are so small that their bite is rarely fatal, as their mouths open at an angle of only about 30 per cent and their teeth are too short to penetrate clothing.

In India the mortality from snake bite is about 29,000 each year, and in the United States it was until recently about 150. In the North, where most snake bites are through the clothing, the mortality was about 10.5 per cent, according to data collected by Dr. P. Willson in the *Archives of Internal Medicine*, in 1907. Until recently it was about 25 per cent in the South and 35 per cent along the Mexican border, according to Dr. Afranio do Amaral.

Snakes increase when their food increases and their enemies decrease. They increase with agriculture, when crops of grain increase the mice and rats on which the snakes feed and their enemies are decreased by man, who kills off the hawks, owls and buzzards, or drives them away. In the Southwest many snakes are killed by the javilina or Mexican wild hog, by the chaparral cock and probably by the badger and skunk, while many young are even killed and eaten by chickens and turkeys.

Their increase can be controlled by destroying their food and hiding places, by clearing up piles of brush and rubbish in which rats and mice live and snakes hide, and having wood piles on elevated stands, with inverted pans to keep the rats, mice and snakes from climbing up. Hogs are useful in destroying snakes in rough, rocky or swampy land where the brush cannot be cleared. Clean out trash under houses and put a fine-meshed wire screen around the bottom to keep the snakes out.

Antivenin has been made to counteract the venom of the bite of the pit-vipers of the United States. It is made by the Antivenin Institute of America at Glenolden, Pennsylvania, at the Division of the Mulford Biological Laboratories. The antivenin is put up in syringes like diphtheria antitoxin and anti-tetanus serum and is made the same way. Each syringe contains 10 c. c. of antivenin, which will neutralize 20 or more mgs. of venom of any American pit-viper *in vivo* and some has been tested to neutralize as high as 53 mgs. *in vitro*. Therefore each syringe should neutralize a minimum lethal dose of venom of a rattlesnake, moccasin or copperhead. We consider the minimum lethal dose for the average man of 150 pounds to be 25 mgs. of venom of a pit-viper. The antivenin is injected subcutaneously in the abdominal region in adults and the gluteal region in children, or it may be injected in the intracostal regions.

*Read in Section on Surgery, Southern Medical Association, Twenty-Second Annual Meeting, Asheville, North Carolina, November 12-15, 1928.

In severe cases it should be given intraperitoneally or intravenously. Antivenin is a polyvalent serum, made from the venom of the American rattlesnakes, moccasins and copperheads. It is made by injecting minute quantities of the mixed venom, into a horse and gradually increasing the amount until the horse can withstand several thousand times the initial dose. By this method it takes about a year to make the antivenin sufficiently strong. Then the dose is increased until the blood has become sufficiently antitoxic. Then nine liters of blood are drawn from the jugular vein of the horse, which gives about four liters of serum, which is further concentrated until it will neutralize 20 or more mgs. of venom per c. c. The serum is then put in a sterilized syringe barrel, with a rubber stopper in the small end, a plunger plug in the wide end, with a piston rod and a glass-capped double-end needle with stylet. It takes only half a minute to assemble the syringe and it is not necessary to sterilize it further. A pearl of iodin, with a swab, is with the kit, to render the site of the injection aseptic. All are placed in a wooden box 4½ inches long by 2 inches in width and 1½ inches deep, with instructions how to use it and with a case report to fill out and return to the Antivenin Institute of America.

The two primary objectives in the treatment of a bite by a pit-viper are:

(1) To prevent the absorption of the venom, so that a lethal dose will not be in the circulation at any time.

(2) To neutralize the venom in the circulation, so that a lethal dose will not be in the circulation at any time.

When the venom of a poisonous snake has been absorbed in the circulation, there is no chemical antidote that can be administered that would destroy the venom in the blood, which would not also destroy the blood, as both are composed of similar colloidal substances.

Consequently, an antivenin for the particular type of snake venom, must be used to neutralize it. Unfortunately most of those bitten by our poisonous snakes are poor people; they cannot afford to buy much, if any, antivenin, so we have never seen a case in which it was the sole treatment. The average Texas diamond-back rattlesnake injects 220 mgs. of venom at a bite or about nine lethal doses; the average cottonmouth moccasin injects 150 mgs. or six lethal doses, and the copperhead 45 mgs. or nearly two lethal doses. Therefore the average adult will require one syringe of antivenin, for each lethal dose, unless some other means are used to delay or decrease the absorption of venom. It has been proved that mechanical suction can withdraw four minimum lethal doses and it should be used, even in cases where antivenin is available, because should a lethal dose of venom get into the circulation, before the antivenin is in circulation, the patient will die. Then again we do not want the venom to be absorbed in the circulation on account of its hemolytic action, for it may destroy so much blood that life cannot continue. We have had cases where the red cells dropped below two million and the hemoglobin to 35 per cent. If one-half or two-thirds of the blood has been destroyed by the venom, it must be replaced, to sustain life. Transfusion would be an ideal method, if we could get the right type of blood and enough of it. In practice we use normal saline solution by subcutaneous injection and Murphy drip. There should be twenty minims of the isotonic saline solution per minute per rectum.

Antivenin is a horse serum, so the usual tests of the patient's susceptibility should be made. Out of over two hundred cases treated with antivenin, we have recorded only one case of susceptibility.

The effect of the action of the antivenin has been to reduce the temperature from 104° F., the pulse from 168, and the respirations from 48 to normal within three hours after injection. It must be remembered, that to obtain the best effects of antivenin, it must be given before the destructive action of the venom has gone too far.

The figures furnished me by the Antivenin Institute of America, Oct. 20, 1928, show that in the calendar year we had 130 bites of poisonous snakes in Texas, of which only 2.7 per cent given antivenin died, while 9 per cent of those not given it died. In the year ending June 1, 1927, we had 150 snake bites recorded in Texas. One hundred and thirty-seven were by rattlesnakes, eight by moccasins and five by copperheads, and the death rate was about 18 per cent. From June 1, 1922, to June 1, 1926, the average death rate of bites of poisonous snakes in Texas was 35 per cent. These were from the figures compiled by the San Antonio Express. We are unable to furnish figures from other states. Some states evidently suppress their figures for certain reasons, usually real estate reasons.

We have to thank the medical profession, the school teachers, the newspapers and the public

for their cooperation in aiding us in the reduction of snake accidents and the fatalities from snake bites.

Discussion follows paper of Dr. Jackson, page 607.

TREATMENT OF SNAKE BITE*

By DUDLEY JACKSON, M.D.,
San Antonio, Texas

This paper is based on the clinical experience gained in treating or supervising the treatment of fifty rattlesnake, copperhead and moccasin bites in and around San Antonio in the last two years. It is also based on a series of 125 experiments conducted by Col. M. L. Crimmins and myself at Dr. Rhea's Veterinary Hospital in San Antonio, Texas. In it are also included the results of experiments conducted at the United States Hygienic Laboratory in Washington by Dr. W. T. Harrison and myself.

For many years the opinion of physicians on the treatment of snake bite has been varied and conflicting. There have been repeated fatalities in spite of many remedies used, and our experiments have shown that most, if not all, of these remedies were without therapeutic value. A large number of people bitten will eventually recover, regardless of the treatment. But there is a small per cent who will die unless carefully treated, and our experiments have been conducted in an effort to take care of these tragic cases.

Our first move was to establish a minimum lethal dose of dry venom for dogs. This was found to be one mg. for each pound of body weight. In each test a control dog was run to check our results. We first tested the value of the magnesium sulphate treatment, magnesium sulphate and glycerine packs being applied externally to a dog which had been given a fatal dose. These were found useless. Venom was then mixed with magnesium sulphate and injected into a dog which died with the usual symptoms. Chloral hydrate treatment was tested with the same results. The old alum and gun powder treatment used by the Indians was found useless; kerosene, which in our community is reputed to be of great value, was used externally without results. Kerosene was then mixed with venom in a test tube, left for 5 minutes, and injected. The dog died as early as his controls.

We carried on a large series of experiments with potassium permanganate, using as solution for injection from a 1-3000 down to a 1 per cent and even pure crystals rubbed in the wound, as suggested by many. This widely heralded remedy was found useless, with the exception of the 1 per cent solution which showed a slightly beneficial effect. However, the drug itself in this solution when injected without the venom, caused serious swelling and sloughing which was almost as bad as the snake bite. The 1-3000 solution which was at this time recommended in the Eighth Corp Army orders, would not even oxidize the venom in a test tube. In these experiments, all of the first aid methods of which we had heard were subjected to the acid test. None was found to be of marked value, and in many instances, as reported in detail in the *Texas State Medical Journal* of July, 1927, our treated animals died at the same time or before the controls.

It has long been taught by the authorities that snake venom is too rapidly absorbed to be mechanically extracted and that it unites with the tissues, producing a new compound which does not lend itself to extraction. Now we conducted about thirteen experiments with suction, injecting dogs with from one lethal dose to as high as ten, and successfully extracted enough venom to save these dogs. Eighty autopsies were done on dogs dying of venom poisoning and from these we were able to publish some interesting facts about the absorption of venom.

It has been generally accepted that venom rapidly absorbs through the venous system and does not remain more than a few minutes in the neighborhood of the bite. Contrary to this, we found that when injected into a dog's thigh, it slowly spreads through the intra-cellular lymph spaces, finds its way into the inguinal glands and there produces a violent hyperemia, followed by the death and necrosis of these glands. It then slowly spreads to the abdominal lymph glands and finally, into the venous system through the thoracic duct. The swelling which progresses up the limb from the area of the bite is none other than dilute venom. Pure fresh snake venom is too violent an irritant to the animal tissue to be absorbed and eliminated as is the case with most non-irritating solutions, and lymph is poured out to dilute it for absorption. Hemolysis occurs, and thus the bloody venom containing fluid of this area is formed. Venom is

*Read in Section on Surgery, Southern Medical Association, Twenty-Second Annual Meeting, Asheville, North Carolina, November 12-15, 1928.

constantly absorbed at the edges of the swelling, but as the concentrated solution spreads, it digests and destroys the tissue with which it comes in contact. If it is allowed to remain in these swollen tissues hemolysis, proteolysis and finally gangrene result. When the swelling of the limb has reached the body and enough venom has been diluted to be absorbed in great quantities, grave systematic symptoms appear such as prostration, convulsions, hematuria, bloody diarrhea, clammy sweat, and respiratory failure.

These facts on the slow absorption of venom point the way to logical treatment, which is free incisions over the area of the bite.

In experiment 19 we injected a dog with 88 mgs. of venom which was freshly milked from live rattlers. Suction treatment was used and the bloody fluid extracted was divided and injected into four dogs. Two dogs died promptly with all the symptoms and autopsy findings of untreated dogs and two had such extensive destruction of tissue that they had to be destroyed. The original dog, which had received four lethal doses, recovered. We also extracted venom from patients bitten by rattlesnakes, one as long as fourteen hours after the bite, injected this into dogs and produced the same pathological condition as was produced by fresh venom, thus proving that the substance extracted was undoubtedly dilute venom.

At the Hygienic Laboratory in Washington a dog was injected with four lethal doses and treatment begun three hours later. The bloody fluid was divided into two containers. To one, 10 c. c. of antivenin were added, and to the other, 10 c. c. of diphtheria antitoxin. These mixtures were set aside at room temperature for one hour and then injected into two dogs. The dog receiving the fluid to which the diphtheria antitoxin had been added died in about seventy-two hours, showing typical lesions of venom poisoning. The dog receiving the fluid to which had been added 10 c. c. of antivenin showed only a slight swelling at the site of the inoculation. This soon disappeared, with no other symptoms. The dog which was originally injected with the four lethal doses, and from which this venom fluid was extracted, recovered. This experiment proved that the substance withdrawn by suction was dilute rattlesnake venom.

In San Antonio hospitals within the last two years we have treated 17 cases of snake bite by suction alone, with no deaths; 13 cases by serum and suction with one death; 10 cases by serum alone with one death; and 5 cases with supportive treatment with no deaths. Five other cases have been treated at adjoining towns. At one time we had nine cases in the hospital. These fifty cases have given us a good clinical knowledge and in addition we have investigated several fatal cases in this section. It is the author's opinion, backed by these clinical findings and by a small series of experimental tests, that the present dose of antivenin which is recommended is entirely inadequate in a serious case of snake poisoning. Col. Crimmins, who has milked thousands of rattlesnakes and who is recognized as the foremost herpetologist in America, contends that the average Texas diamond-back rattlesnake can inject in one bite an average of 220 mgs. of venom. From our experiments and other sources of information, it is quite evident that one syringe of antivenin will neutralize only about 10 mgs. of venom in the animal body. Therefore, if in treating these fatal cases with antivenin alone, especially children, you follow the present teachings as to doses, fatalities will continue. A child weighing sixty pounds can neutralize within itself about 10 mg. of venom, and one syringe of antivenin will neutralize 10 mg. To be extremely conservative, we will assume that only 150 mg. have been injected. Subtract 20 mg. neutralized by the child and the antivenin and you have at least 130 mg. left to destroy your patient. In these cases it would take at least 13 ampoules of antivenin if this were given alone. We have extracted as much as 100 mg. of rattlesnake venom from a dog by mechanical suction, so the advantage of using suction in the treatment of these cases can readily be seen.

Antivenin is a useful remedy and is the only known substance, according to Col. Crimmins, that will neutralize the venom when once it is absorbed, and it is impossible to prevent some absorption, regardless of how carefully you use suction. Accordingly, the ideal treatment and the one which has yielded us the best results is as follows:

Release all tight tourniquets and replace them by one just tight enough to obstruct the lymph circulation and not the free venous return. Under novocain make a large cross cut at the fang marks. Make a circle of secondary cross cuts ¼ by ¼ inch about three inches from the original wound. In the small incisions, inject several hundred c. c. of a 1 per cent salt solution and apply suction over the original cut to wash out dilute venom. This is continued for about one-half to one hour. If a vein is cut the hemorrhage is controlled, as it is bloody serum producing the

swelling, and blood must be conserved. Once every hour for from 10 to 15 hours suction is repeated for about twenty minutes, using cupping over first one incision and then the other. A small rubber bulb with inverted glass funnel is usually used. However, Biers' hyperemic cup or suction machine used in tonsillectomy with an attached ear speculum is found useful. Quite a quantity of venom is diluted each hour and can be extracted. Fluid also leaks from the wound between extractions. If the swelling progresses, a bracelet of incisions is made around the highest point of the swelling, and new incisions are made where this fluid has collected in large quantities. In most cases treated by others, we have found that an insufficient number of incisions was made and suction was not continued for a sufficient length of time. Morphine or paregoric is given for pain, stimulants when indicated, hypodermoclysis and blood transfusions if the case comes to the physician late and shows extensive destruction of red cells with count below 3,000,000. The hemoglobin and red blood cell counts are made every three hours, a saline cathartic is given and colonic irrigations of salt and soda once every four hours. Between treatments, the limb is kept wrapped in hot fomentations, either bichloride of mercury (1 to 10,000) and magnesium sulphate, or sodium citrate solution, to increase the outflow of venomous serum and help keep the wound from being infected.

It is believed that strict adherence to this outline of treatment will result in the saving of many lives that would be listed otherwise as cases of fatal snake bite.

DISCUSSION (*Abstract*)

Papers of Col. Crimmins and Dr. Jackson.

Dr. Robert A. Strong, Hagerstown, Md.—The progress which has been made has placed the subject of ophiology on a much firmer foundation than was ever possible in the past when it was based almost entirely on the misinformation of reptilian folklore and fable. I am quite sure, therefore, that much of what you have heard today has been a revelation to those of you who were not aware that the subject has been receiving serious attention.

It has been more than twenty-five years since Calmette immunized horses against cobra venom, but the first effective therapeutic serum was developed at the Institute of Serum Therapy of Butantan, Sao Paulo, Brazil. For this reason when the Antivenin Institute of America and Harvard University became interested in developing a serum for the snakes of North America they naturally sought the assistance of the Director of the South American institute. Soft of voice, suave in manner and talking with an accent suggestive of old Castile, this modern St. Patrick crossed the equator and gave us freely the information he had acquired through years of experience in a country where venomous snakes were a serious problem. He was Dr. Afranio do Amaral. After a complete survey of the United States was made it was found that venomous serpents were distributed all over it, except in the extreme northeastern portion. It was also found that the rattlesnake, moccasin and copperhead were the varieties which were the chief offenders. Moreover, the increase in automobile touring, hiking, camping and outdoor living had increased the hazard and had emphasized the need for a polyvalent serum which would neutralize effectively the venom of these species. Such a serum was produced and is now available in an adequate quantity, so there is no reason why a person bitten by any of the three varieties mentioned should receive any treatment other than that which you have heard today. Any other treatment is as useless as were the treatments of diphtheria before von Behring gave us anti-diphtheritic serum.

A vast amount of orderly and scientific information is available and I commend it to your serious consideration. The misinformation about snakes which has existed and still exists should be overcome. Boy Scouts should receive accurate instruction. In fact, all who are interested in outdoor living are entitled to the real facts about snakes. An example of the disastrous results of misinformation came to my attention a short while ago. A young Boy Scout in Louisiana, who had received instructions that venomous snakes always had wide, triangular heads, a pit between the nose and eyes, and the other marks that are characteristic, found a coral snake which happens to be extremely venomous, but does not have the conventional marks. He considered it perfectly safe to adopt this beautifully-colored snake. After possessing it a day the snake nipped his chest while he had him in the bosom of his shirt. The boy died in three hours. There is no serum for this variety, because it is a very rare species, but accurate instruction would have forewarned the boy. The importance of orderly information is obvious, and it is not hard to acquire.

Dr. R. H. Hutchison, Philadelphia, Pa.—As Secretary of the Antivenin Institute of America, I have been gathering and analyzing reports of snake-bite poisoning from all parts of the Country. We have also undertaken recently in our laboratories experimental investigations along lines somewhat similar to those of Dr. Jackson. This work has been done by Dr. Thomas S. Githens and Mr. R. E. Stadelman, and it may add interest to Dr. Jackson's paper to indicate briefly some of the lines along which this work is developing.

First, let me say that we are pleased to note that Dr. Jackson relegates to their proper place among the worthless measures many old-fashioned popular remedies and shows the entire lack of value of kerosene oil, turpentine, the application of a split chicken, burning gunpowder in the wound, and the like. Even potassium permanganate is discarded, thus confirming the findings of others.

It must be conceded that Dr. Jackson has done a real service in making explicit what has heretofore been implicit regarding incision and suction in the treatment of snake-bite. Incision, and sometimes incision with suction, have been used by various peoples. You probably know that Indian tribes have used some such

procedure. Some interesting records from colonial times in Massachusetts show that it was used by the early settlers there. Dr. Jackson's contribution to the subject consists primarily in demonstrating that by this method highly toxic material is withdrawn.

Our experimental work is also being done with dogs. We have found that dogs are exceedingly variable in their resistance to snake venoms. For example, it has happened that one dog receiving 1 mg. per pound died, while another receiving 2.5 mg. per pound recovered without treatment. This indicates how confusing the results of dog experiments may be and how guarded we must be in drawing conclusions from them.

A large snake, if it makes a good strike, will make an intramuscular injection of venom. Our experiments have been based on this consideration. Measured quantities of venom have been injected one-half inch deep into the thigh muscles. Within a few minutes a single incision was made to a like depth at the same site and suction applied. We have confirmed the finding that the fluid removed is highly toxic and that the toxicity can be neutralized by antivenin, but we have not been able thus far to demonstrate the life-saving value of the procedure following intramuscular injection.

Another series of observations raises in our minds a question as to the time factor in the application of suction. One cubic centimeter of the toxic fluid withdrawn during the first half hour after venom injection killed pigeons within thirty minutes. At each succeeding suction period the fluid withdrawn became less toxic and 1 c. c. of that obtained after five hours had no apparent effect on pigeons. The question we raise is whether mechanical suction in practical application has real value after the first two or three hours.

Dr. Jackson's references to antivenin prompt mention of another line along which we are directing some experimental work. This has to do with the influence of the method of administration on the effectiveness of the serum. It is already indicated that antivenin given intravenously shows a greater neutralizing action than when it is given either intramuscularly or subcutaneously.

Dr. Evarts A. Graham, St. Louis, Mo.—I should like to know whether the commercial antivenin is effective against all snake bites, or is it specific only for some particular varieties of snakes?

Dr. Jackson (closing).—The material that was sucked out of the dogs was said to be very lethal, but this effect to decrease as time goes on. That is true, but if the wound is not cut open and suction is not applied, this does not hold good. If one waits for fourteen hours and then extracts enough venom to inject into a dog, it will kill just as quickly as that first secured by suction.

Col. Crimmins (closing).—Replying to Dr. Graham, the commercial product is a polyvalent serum that is specific against eleven varieties of rattlesnakes, the moccasin and copperhead. It is not specific against the coral snake, for we found that the coral rarely bites a person, and we have felt that it was not necessary to make a special serum for it.

STUDIES ON ASCARIASIS IN VIRGINIA*†
A PRELIMINARY REPORT

By W. W. Cort, Ph.D.,
G. F. Otto, Sc.D.,
and
L. A. Spindler, Sc.D.,
Baltimore, Md.

The human ascarid, *Ascaris lumbricoides*, is probably the most widely distributed and frequent of all the parasitic worms of man. The remarkable resistance of its eggs gives this parasite a much wider range of distribution than the hookworm in regions with temperate climate and low rainfall. Egg production is almost unbelievably great, it having been demonstrated that a single female may give off over 200,000 eggs per day.[1] [2] These eggs are in the single cell stage and require a period of development outside of the body of the host of about three weeks or more, depending on environmental conditions, before the embryos reach the infective stage. This means that human infection can never be a matter of direct fecal contamination as in the case of the pinworm, but that it must come from soil or water previously polluted which may at the time show no trace whatever of fecal material. Infection with ascaris comes about by the entrance of the embryonated egg into the mouth on soiled fingers or with food or water. After entrance the eggs normally pass through the stomach and hatch in the small intestine. The newly hatched larvae then penetrate the intestinal wall, enter the circulatory system and are carried to the lungs. Here they break out from the capillaries into the air sacs and make their way back to the intestine by way of the respiratory and digestive tracts. Although the migrating larvae may produce minute lesions in their passage through the liver, and have occa-

*Read in Section on Public Health, Southern Medical Association, Twenty-Second Annual Meeting, Asheville, North Carolina, November 12-15, 1928.

†From the Department of Helminthology of the School of Hygiene and Public Health of the Johns Hopkins University. This study is part of the program of researches on ascariasis in children which is being conducted under the auspices of the National Research Council with the aid of a grant from the American Child Health Association. This program, which is for five years' work, got under way in September, 1927. In connection with it we are trying to get together all the information we possibly can on the distribution and importance in disease of human ascaris in the United States. We would, therefore, be very grateful for information on this subject from public health men or practicing physicians.

sionally been found in the kidneys, spleen, lymph nodes, thyroid gland, brain, and other organs, by far the chief injury that they produce is from the hemorrhages which occur in the lungs when they break from the capillaries to the air sacs. After reaching the intestine following the lung migration the young ascarids increase greatly in size and reach sexual maturity in about six weeks. The exact relation of this migration of ascaris to respiratory diseases and the extent to which the presence of these parasites affects the general health of the population groups in which they occur, are matters which are not at the present time entirely clear and on which further investigation is badly needed.

Occasional cases of human ascaris have been reported from all parts of the United States. In most sections of the country, however, there is available no evidence of the presence of even small endemic centers, where any considerable percentage of the population is infested. Many parts of the southern United States, with the greatest hookworm infestation, have a surprisingly low ascaris incidence. F. C. and E. L. Caldwell[3] give a summary of statistics for Alabama which shows an ascaris incidence of 0.2 per cent in the lower coastal plain, where hookworm after years of control work is still in many places an important public health problem. Similar information is available for Florida, eastern Virginia, and other districts where hookworm was and in many places still is very prevalent. In fact the data at present available indicate that the gulf states and the south Atlantic seaboard have a surprisingly low incidence of ascaris, and that it is only in the mountainous regions of the southeastern United States, especially in Virginia, North Carolina, Kentucky and Tennessee, that infestation with this parasite is widespread and heavy. No explanation can at present be offered for this peculiar distribution. Certainly nowhere else in the world, as far as information is available, does this parasite show any special predilection for mountain regions, and the insanitary conditions necessary for its spread occur to just as great an extent in some of the areas of the South where the incidence is low.

Wise County, Virginia, where we carried on our investigations on intestinal parasites during the past summer, is in the heart of the mountains next to the Kentucky border.* It has a population of about 50,000 engaged in mining and agriculture. The workers in the mines are not foreigners brought in from outside, as in mining regions further to the North, but are recruited from the indigenous population. One of the striking features is the small proportion of negroes, who make up less than 10 per cent of the population. The county is very mountainous, so that except in a few valleys the farms are very small. The development of mining has produced several towns of 2000 to 5000 inhabitants in which some of the miners live. Most of the mine workers, however, are gathered together into small camps which have been developed by the companies near the sites of the mines. In addition to the studies in Wise County we also made more limited surveys in Russell and Smyth Counties.

The methods used in these investigations were similar to those developed in the last few years in making epidemiologic studies on hookworm disease. To give quantitative information on the distribution and intensity of the infestation, the dilution egg count developed by Stoll,[4 5] was utilized. Egg counts for all the intestinal worms were made on over 2000 different individuals from groups which would be most likely to harbor intestinal parasites and which represented the different types of conditions for the general region. After a preliminary analysis of our egg counts for any group, the next step was to inspect carefully typical households to see what conditions were present to account for the particular kind of worm infestation shown by the egg count series. By studying the soil pollution habits of a number of families heavily infested with ascaris and comparing them with those which were negative or lightly infested, it has been possible to obtain information on the factors in the environment and the habits of the people that are favorable for the spread of this parasite. In many cases where the infestation of the people could not be explained by the finding of traces of soil pollution, samples of soil were taken from suspected places near the houses and examined for the presence of ascaris eggs by a method similar to that used by Caldwell

*These investigations were made possible by the interest and cooperation of the officials of the State Board of Health of Virginia and of the Wise County Health Department. Dr. W. R. Culbertson, Director of the Wise County Health Department, entered enthusiastically into the work himself and placed the facilities of his department at our command. We wish also to express our appreciation to Dr. C. W. Scott, Mr. H. L. Freese, Mr. C. B. Neblitt, Mr. J. F. Ward, Miss Grace Hale, and Miss Ruby Revere for assistance in the field work.

and Caldwell.*[3] These examinations of the soil give at places where soil pollution is evident information on the favorableness or unfavorableness of the situation for the development of the eggs. They also make it possible in certain situations to get evidence of sources of infestation in places where no signs of soil pollution were found at the time of the inspection.

In the present paper we will bring forward in summarized form the data gathered during our summer researches on the distribution, methods of spread and significance of ascaris in this region. These findings will be made the basis for a discussion of certain of the problems which the health officer has to face in backward rural communities in preventing soil pollution and in developing a satisfactory sanitary sense in the people.

ANALYSIS OF THE EGG COUNTS OF ASCARIS FOR THE COMMUNITIES STUDIED

Egg counts were made for 2152 different individuals, representing for the most part the poorest, most backward groups of the communities studied.† A considerable excess of children, especially those of school age, were examined, although the numbers of the adults and youngest children are sufficient to give significant information.

The incidence of *Ascaris lumbricoides* in the total series was 48.4 per cent. Both the incidence and the degree of infestation as measured by the average egg count and the estimated average worm burden is about the same in the males and females. The incidence in children is about twice that of the adults (about 60 per cent as compared with 30 per cent), and the intensity of infestation is several times greater. It is especially significant that in the positive cases taken alone the average worm burden of the children is about three times that of adults. In fact, the intensity of the ascaris infestation is already very high in the pre-school group, reaches its peak at about eight years, and begins to fall off rapidly after 12 years. The burden of this parasite, therefore, falls particularly on young children at the age when any handicap is most serious.

Over 80 per cent of the total examinations were from Wise County. The others which were from Russell and Smyth Counties showed that heavy ascaris infestation was also present in widely scattered places in both these two counties. This fact and the information from earlier examinations which shows a high incidence in Dickenson, Lee and Wythe Counties indicate that this parasite is widespread in southwestern Virginia. In fact, on account of the extensive sanitary work carried on in Wise County, it seems very probable that conditions in many places in the general region are worse than those we found in this County. Also the records available from the mountain sections of Kentucky, Tennessee and North Carolina suggest that ascaris infestation of this same type is also widespread in these regions.

The Wise County studies include both small mining camps and other similar communities and isolated families of the farmers and mountaineers from all parts of the County. No significant differences in these two types of situations were found either in the degree of ascaris infestation or in the factors which influence its spread. In some cases extraordinarily heavy worm burdens had been built up in isolated families and on the other hand some of our worst groups were on the outskirts of the larger towns. In most of the people in the larger towns, however, the standards of life and sanitation are such that infestation with intestinal parasites is rare.

Since most of the families included in our egg count series had made some attempt at household sanitation in response to the efforts of the County Health Department since 1921, the heavy ascaris infestation found at homes for the most part provided with privies might appear at first sight to be rather discouraging. On the other hand, there are present in the rural districts and even in some of the poorest community groups a number of houses, in many cases the majority, where the privies are kept up and used and where the people by their own efforts at household sanitation are entirely or almost entirely protected from intestinal parasites. Another encouraging feature is that the majority of the people in the mining camps where the

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*Our most recent development of this method differs somewhat from the Caldwell technic. About 10 c. c. of 30 per cent antiformin are added to about 10 grams of the soil in a 50 c. c. centrifuge tube, mixed thoroughly, and left standing for about an hour. The tubes are then filled almost full with a sodium dichromate solution (Na_2-Cr_2-O_7) having a specific gravity of 1.35. After their contents are mixed thoroughly the tubes are placed in a centrifuge and run at a moderate speed for a few minutes. Loopings of the surface film can then be examined for the presence of the eggs and the stages of development of any eggs found can be noted. We owe the developments in this method chiefly to Dr. H. W. Brown.

†The particular groups for investigation were chosen on the advice of Dr. W. R. Culbertson. He stated that in his opinion the series examined from Wise County was a fairly good cross section of the poorest two-thirds of the county population.

companies provide really adequate sanitary facilities used the privies and were comparatively free from intestinal parasites. Further, in many cases people of the poorest, most ignorant and degraded type protected themselves by using their privies which in some cases were in the last stages of dilapidation. On the other hand, quite a number of families were found which appeared to be fairly well off and were clean and decent in other respects, but in whom on account of soil pollution habits of certain individuals, heavy ascaris infestation was found.

No racial differences between negroes and whites in susceptibility to ascaris were apparent, for under like conditions of soil pollution similar worm burdens were present. Some of the negroes of the region had responded very satisfactorily to the efforts of the health department. For example, in a small negro community on the edge of one of the towns practically no soil pollution, even by the youngest children, was found, and the people were almost entirely free from ascaris. Some of these people lived in very poor shacks and were about as dirty as they possibly could be. This group contrasted with a near-by mining camp inhabited entirely by white people which, in spite of the presence of privies, showed a general soil pollution, especially by the children, and a very high ascaris level. Another example of this same thing was found in one of the best mining camps of the region where splendid sanitary facilities were provided and where about half the camp was given over to negro families. Not a single negro family in this place was included in those which on account of soil pollution in the yards had heavy ascaris infestation.

FACTORS INFLUENCING THE DISSEMINATION OF ASCARIS IN SOUTHWESTERN VIRGINIA

With information from the egg counts at hand, inspections for soil pollution supplemented by the examination of the soil for parasite eggs were made of families living under different types of conditions who showed heavy ascaris infestation. Such families which we have come to call ascaris families were compared with others that were lightly infested or negative.

The most striking point that comes out of the soil pollution surveys was that except in a comparatively few cases the adults used the privies and almost all of the soil pollution was from the children, especially those of pre-school and early school age, in which group, it will be recalled, was found the heaviest ascaris infestation.

Further, the individual household and not any community group was almost always the unit in relation to ascaris dissemination. Sources of infection common to groups of families or to a part or the whole of a community were so rare that examples of them may be of interest. In one village large numbers of stools were found on the floor and underneath an old deserted church which served as a play house for young children. Infective eggs were present in both places in such numbers as to suggest that here was an important general source of infection for the children of the neighborhood. In a few cases also potential sources of community infection were found in school yards. We also in a few instances found that children from well sanitated families were evidently picking up ascaris eggs from playing with the children at houses where conditions were bad. In one area three families, above the general economic level and living close together, illustrated this point very clearly. In one of these families the children polluted the soil in the yard and had fairly heavy ascaris infestations. The children from one of the other two families where no soil pollution at all was found were allowed to play with the ascaris family and were infested, while the other family in which the children were kept strictly at home was free from this parasite. Another example of this same thing was from a well sanitated mining camp where the children on both sides of a double house had heavy infestations when soil pollution was found on only one side, the other family giving every indication of care in household sanitation.

Since the spread of ascaris is often attributed to the water supply, as in Chandler's researches in India, we investigated for each household surveyed the source of the water used. At most of the rural houses the water was obtained from springs usually at a considerable distance. In not a single case did we find soil pollution near these springs or in places that drained directly into them. In fact the analysis of the spring water in some of the most heavily infested communities gave no evidence whatever of fecal pollution. We have come to the conclusion, therefore, that infestation from the water supply can hardly be an important factor in any of the groups studied.

In fact, the one feature which was found to be common to practically all the ascaris families was the failure of the younger children to use the privies and their pollution of the soil close to the houses. At such places stools were found along the sides of the house, next to the porches

or door steps, by the side of the sheds, around the privies, and even under the houses. Many of the houses built on hillsides were raised from the ground, leaving considerable space under the floor. Here the children often played, especially in rainy weather, and here they deposited their stools, seeding the soil with ascaris eggs which developed rapidly to the infective stage in the shady, moist environment. No other type of soil pollution was so common, and no other situations yielded a larger proportion of infective eggs in the soil samples examined. At houses of ascaris families soil examinations often showed the presence of eggs in paths, in the front yards, or even inside the houses themselves, indicating a wide scattering. It was not uncommon to find ascaris houses where no evidence of soil pollution could be found, but where the soil samples yielded numbers of eggs. In some of these places the fact that practically all the eggs found were embryonated suggested that they had persisted from an earlier period of soil pollution, and in others the absence of stools could be attributed to the presence of large numbers of chickens. It is of interest that in practically every house where heavy ascaris infestation was present, either soil pollution or embryonated eggs were found in the yard close to the house. It would take too long to mention all the various types of soil pollution found. In some places the overflow from the privies had infected the soil where children played and in others barns and outhouses were involved. Throughout the whole region, however, the outstanding feature is the soil pollution near the houses in a few cases by whole families, but in the great majority of cases by the younger children only. In these mountain regions, therefore, where the preliminary steps in household sanitation have been taken, ascaris infestation becomes to a very considerable extent an index of the use of the privies by the children.

SUGGESTIONS FOR CONTROL

Suggestions for control of ascaris, therefore, must take into consideration not alone the introduction of privies in insanitated districts, but their conservation, replacement and use by children where the preliminary sanitation has been accomplished. These are the outstanding problems of household sanitation in the rural districts of the South and any suggestions we can make can hardly help but be rather obvious and trite. One very practical point seems, in many places at least, to have been overlooked. That is the mechanical adaptation of the privy for children's use. In other words, why not have a children's hole? In many places the mothers definitely stated that the little children did not use the privies because it was so difficult for them to get up on the seats and also because they were afraid of falling in. If to control ascaris we must teach young children not to pollute the soil, certainly the first step must be the modification of the privies so that they can conveniently and safely use them.

With this primary step taken, the problem becomes chiefly one of intensive education in which the health officer, sanitary inspector, public health nurse, and school teacher all can have a part. Such instruction is a part of the general program of education of backward communities in the general principles of proper living. Long experience has taught that it is only by the constant hammering away over long periods of time that habits of long standing can be changed. Here, as in every other educational movement, the best chance is to "catch them young," and the best place to reach the youngsters is in the public schools. This brings us face to face with the problem of whether the rural school, in such communities as we have been discussing, is really doing its part in solving these problems. Would it not be possible to plan for rural districts in regions where even the simplest principles of sanitation are constantly violated, a course of more intensive instruction to supplement that now given in the general hygiene courses? What can possibly be given the child which is of more importance than repeated instruction from the early grades onward, by teachers who are themselves adequately instructed, in those fundamental principles of proper living which will protect him and those associated with him from the long series of filth-borne disease? Would it not be possible to adapt more adequately the subject matter of the hygiene courses to the particular locality? Instruction that is of great importance for rural children in backward sections certainly has no place in city schools, and just as surely a great deal of material appropriate for the cities is entirely meaningless in country districts. No text book in hygiene can be written which used alone will be appropriate for the great variety of conditions found in any one state. Would it not be possible to prepare for use where needed small text books of the type of the readers used in early grades which would cover in a simple but full enough manner the problems of personal hygiene and household sanitation for country families of the poorer, more backward type? Of necessity the develop-

ment of such courses will require the most complete cooperation between health authorities and the educational system and present difficult problems in the adaptation of subject matter to local conditions and in the adequate training of teachers. But difficult as the problems are, none of them seems insoluble. If, as many claim, prohibition was brought about by teaching the dangers of alcohol to the school children of the past generation, why could not ascaris be eliminated from the mountains of the southeastern United States by intensive instruction in the early grades in the dangers of soil pollution?

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DISCUSSION (*Abstract*)

Dr. Roy K. Flannagan, Richmond, Va.—We are very fortunate indeed in Virginia in having these studies conducted in one of our mountain counties; fortunate, too, in having a good working organization there to back Dr. Cort up and assist him in the studies. The thing that has struck me most forcibly about Dr. Cort in his work is that he has comprehended fully the public health significance of what he is doing.

It appears clear from these investigations that, though we have built an enormous number of privies, we have paid little, if any, attention to the needs of the little pre-school child. We have not made a small enough hole for him. We have been enlarging the capacity of our privy pits and making them more and more dangerous for the little folks to use. Wasn't it James Whitcomb Riley who said: "He jack-knifed down through the seat?" It is not an impossible situation. I have been told that some children have been drowned in septic privies. Following Dr. Cort's investigations, it will be necessary to pay more attention to the privy comfort and convenience of the young children on the place so that soil pollution and ascaris infestation by them will not be inevitable.

I was with Dr. Cort in a number of investigations, and we ran on one village where the houses were built on a steep slope. There was a street in front of some of the houses, but the houses on one side of the street were all on stilts, as it were, facing the road, and the others on stilts at the back. The children of the household made playhouses under each house, and it depended altogether upon the general social status or cleanly habits of the family as to how badly the under side of the house was infested with ascaris worms. In many of the places the ground was apparently clean, even looked as if it had been swept, and yet, by taking a trowel and scooping up the dirt and examining it under the microscope, ascaris eggs could be found in abundance, showing that soil pollution by the children was habitual.

Perhaps many of you have heard of the experiment in ascaris infection by two Japanese students who decided to test out the actual danger to themselves of infection by ascaris. One swallowed 1000 eggs and waited the result, and as symptoms appeared recorded them. The other swallowed 3000 eggs. The man with 1000 managed to keep his mentality working sufficient to keep the record until he recovered; the other man promptly became delirious and he developed a very intensive pneumonia due to the transmigration of the larvae through his lungs. If ascaris pneumonia can thus be caused experimentally, surely in heavily infested neighborhoods there is reason to believe that it may at times occur. Every physician has had puzzling cases of summertime pneumonia in children. Perhaps ascaris infestations may explain some of them.

There is one other point that Dr. Cort's work suggests to my mind. We have all through the Country many public health nurses. These must be shown the necessity of seeing that the little children at the homes they visit are not permitted to defecate on the ground immediately around the house. By working on the mothers surprising results in rural child health can be achieved.

Dr. E. L. Bishop, Nashville, Tenn.—As Dr. Flannagan brought out, we are to be congratulated on having a research worker in this field who has the practical point of view. It is largely for that reason that we have urged Dr. Cort to extend his research work to Tennessee next year. We believe that less is known about the practical epidemiological aspect of ascaris than about almost anything else as widely spread as is ascaris. We know little about its clinical picture and little about its effect on the population, and I believe it is our obligation to engage in that study which will answer the question, if it is answerable. We propose next summer to institute some investigations of this particular problem in Tennessee, with Dr. Cort directing us and with the Department of Preventive Medicine of Vanderbilt University assisting, and to carry on the work after Dr. Cort is through with his particular part of the study. It is to be a continuous study until we have found the answer.

We expect to study the geographic distribution, and are at present having analyzed our past experience. We have some 100,000 examinations of persons which have not been completely studied. We shall have them analyzed by age, sex and race. That will be purely a preliminary as a measure in determining the areas for more intensive study later on. Then we wish to study further the clinical aspects and epidemiology of ascaris, and to engage the cooperation of Vanderbilt University's Department of Medicine, as well as the Department of Preventive Medicine. I remember having had one case of pneumonia which was very definitely complicated with ascaris.

Also we wish to study the reinfection rate: for example, to study an area where there is full-time health work and another where nothing is being done and compare the rate.

Dr. A. T. McCormack, Louisville, Ky.—During our hookworm survey we examined 450,000 people in Kentucky at random, not that we suspected them of having any parasitic infection. Thirty-four per cent

had ascaris infection and 33 per cent had hookworm infection at that time.

Since then we have completed the study of some 250,000 additional individuals, most of them suspected of having some infection. It has been extremely interesting to us that the hookworm percentage has constantly decreased, while the ascariasis has remained stationary.

Recently in Leslie and adjacent counties three or four well trained mid-wife nurses conducted health examinations. They found very early that one of their most serious problems was ascaris infection.

Dr. Henry E. Meleney, Nashville, Tenn.—There are comparatively few references in the literature to clinical ascariasis and it is difficult to separate the symptoms due to this parasite from those of other infections in the patient. We need as many observations as possible on this subject, and will appreciate it if any interested physicians will report to us during the next year or two any clinical manifestations of ascariasis which they may encounter, due either to the larvae passing through the lung, or to the presence of the adult worms in the intestine. Such observations may be addressed either to Dr. E. L. Bishop, Tennessee State Commissioner of Health, or to me, at the School of Medicine, Vanderbilt University. We shall, of course, give due credit for any such observations.

One of the most important points which Dr. Cort has made is the necessity of teaching the school children in any community how to avoid the infections present in that community, rather than of merely giving them the general principles of public health. I hope the health officers in the various states concerned in this problem will bring pressure to bear on Dr. Cort to write a syllabus for use in schools on the prevention of ascaris infestation.

Dr. Felix J. Underwood, Jackson, Miss.—We must not only induce the country people to construct privies, but also teach each member of the family to use them. Along with this investigational work with the children, some attention should be given to the hogs as a spreader of ascaris, for we know that around many of the poorer homes in the rural districts hogs are to be found very near the house, and sometimes they roam all over the premises.

Dr. Cort (closing).—I shall try to answer first the question asked by Dr. Underwood in regard to the possible role of the pig in spreading ascaris to man. In the area where we worked last summer there were very few pigs, so that they were not a complicating factor in our studies. We hope later in our field work to have an opportunity to investigate the problem of the relation of the pig to human ascaris.

In closing I wish to request the cooperation of the health officers in regions where ascariasis is endemic in obtaining information on its prevalence and importance as a factor in the health of children. We are undertaking a five-year program of research on ascaris in the United States and as one of the phases of this program we wish to gather together all the information that we possibly can on the distribution of this parasite in different parts of the Country. We should, therefore, be very much pleased to receive any unpublished data on the incidence of ascaris which might be obtained from routine fecal examinations, or any information whatever on its presence or importance in particular localities.

CLINICAL INVESTIGATION OF PYELITIS*

By Roy P. Finney, M.D.,
Spartanburg, S. C.

It is the consensus of urologic opinion that there are three possible routes through which infection may reach the kidney: first, the ascending or urogenous route; second, the descending or hematogenous route; third, by traversing the lymph channels either from the intestine to the kidney, or from the pelvic organs to the kidney, which has been called the lymphogenous route. Of the three it seems likely that this is of the least importance, though Francke,[1] by injecting the lymphatics of the large bowel, believes that he has demonstrated a direct lymphatic communication between it and the kidney. And Sweet and Stewart,[2] from experimental work done on the lymphatic network of the bladder and ureters, conclude that infection may travel along it upward to the kidneys. Some clinical facts support the views of both observers. Thus Kretschmer and others have noted gastro-intestinal disorders in a large number of their patients with infected kidneys, and it is common belief that chronic constipation predisposes to pyelitis. However, other considerations militate against the probability of the lymphatic route's being a frequent one.

The idea of hematogenous infection was an outgrowth of the general acceptance of the important doctrine of focal infection. It received great impetus from the experimental work of Bumpus and Meisser,[3] who in 1921 reported that streptococci found in the teeth and tonsils of patients affected with pyelitis had a selective action on the kidneys of rabbits when injected intravenously. They concluded that pyelitis is usually of hematogenous origin, that the streptococcus is the infecting organism and the colon bacillus a secondary invader which outgrows it and usurps the entire field.

Other clinical and experimental work tends to support this view, so that it has gained wide acceptance by the urological profession. Thus Widal and Bernard[4] and Panton and Tidy[5] report positive blood cultures in a few cases of pyelitis. Cecil[6] in 1924 concluded that "infection reaches the kidney through the blood stream and not by ascending the ureter except in rare instances." Hundley,[7] in an excellent review of the subject, concluded that pyelitis is most

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frequently of hematogenous origin, the infection beginning in the parenchyma and extending downward to the pelvis.

Not a few observers feel that while it is important, the hematogenous route of renal invasion is being over-emphasized; that it may be most unwise to minimize the importance of ascending infection and to close our eyes to some established facts and suggestive experiments. For example, the relative preponderance of pyelitis in the female cannot be denied. In 2000 cases which I recently collected from the literature females were affected in 76 per cent. (Here I wish to stress the point that statistics on pyelitis as to sex are much more valuable when coming from the general practitioner for the reason that his clientele is composed of males and females in approximately equal numbers. Few urologists can fulfill this requirement.) The most logical explanation for this feminine majority appears to be the old one that the female urethra is a less effective barrier to bacteria for the following anatomical reasons: first, it is shorter than the male urethra; second, it lacks the strong sphincters of the male urethra; third, it is in close proximity to the rectum and is immediately adjacent to the vagina which continually swarms with a variety of bacteria.

Another suggestive clinical phenomenon is that of the "catheter chill." To explain it by saying that the mere passage of a dirty catheter produces trauma to the extent of introducing organisms either into the lymph or blood circulation seems rather far-fetched.

Again there is a distinct difference between an uncomplicated primary acute pyelitis and pyelitis occurring as a complication of stone or other obstructive uropathy. In the latter, fever and toxemia are likely to precede bladder symptoms in a majority of the cases; while in the former, dysuria, mild or severe, usually precedes the onset of high temperature, toxemia and pain in the loin. In 500 cases of uncomplicated pyelitis reported by various authors, dysuria preceded actual illness in 69 per cent. However, it is often transitory; indeed in my experience it is most unusual for severe bladder pain and strangury to complicate acute pyelitis so long as high fever is present, even though the urine be highly concentrated and thick with pus and bacteria. Such experiences cast doubt over the theory that such vesical irritability is often due to the irritative quality of the urine discharged from the infected kidney, or to referred pain from the kidney or ureter. Cystitis of some degree is present, and the explanation that it is primary and pyelitis secondary, rather than the reverse, is probably true more often than most of us admit.

From the experimental standpoint, Helmholz showed that lesions of the kidneys could be produced in rabbits by the simple introduction of colon bacilli into the bladder without producing either trauma or obstruction. And Helmholz and Millikin[8] studied the etiology of pyelitis complicating upper respiratory infections, and conclude that:

"It is not caused by bacteria producing the primary infection, but almost invariably by colon bacilli. In four cases of pyelitis secondary to upper respiratory infection the organisms causing the primary infection did not reveal a tendency to localize in the kidney when injected intravenously in rabbits."

The phenomenon of ureteral regurgitation must certainly be numbered among the most interesting and important recent urologic discoveries. It was produced experimentally in rabbits by Wislocki and O'Connor[9] and later was demonstrated in the human by means of the x-ray by Kretschmer and others. It occurs, of course, only when there is an insufficiency of the uretero-vesical valve through over-distention of the bladder, local disease involving the valve, or because of some type of neuromotor dysfunction. It has been considered an infrequent and relatively harmless occurrence by most observers.

In March, 1928, I had the unexpected pleasure of observing ureteral regurgitation by means of the cystoscope in the following cases:

Case 1.—T. J. J., a white man, age 68, consulted me complaining of inability to void freely and of hematuria. For the past year he had noticed an increasing difficulty in starting the urinary stream. Completion of the act of micturition was frequently accompanied by strangury and a little terminal blood would appear. Nicturia occurred six to ten times. The family and past history were of no importance.

On examination, the bladder was visibly distended and 28 ounces of urine were withdrawn per catheter, which entered the bladder without difficulty. The prostate was only moderately enlarged. Cysto-urethroscopic examination, using the McCarthy pan-endoscope, revealed an extensive carcinoma involving the left lateral wall of the bladder and extending downward into the posterior urethra. The ureteral orifices were not overgrown by the tumor and appeared normal. The case was considered inoperable and electro-coagulation was advised and accepted. It was carried out at ten-day intervals and at the end of the third sitting all of the tumor within the bladder had been destroyed. Thus far there had been no reaction following the burnings and his general condition was improving rapidly. At the fourth sitting, before attacking that part of the growth within the urethra, he was given 40 mg. of indigo-carmine intravenously to determine the kidney function by chromo-cystoscopy. The blue color appeared at both ureteral orifices in four and a half minutes and in six minutes was being expelled in strong,

large, deep blue spurts at the rate of four per minute, the ureteral lips closing normally in the intervals. The current was turned on and the electrode applied to the growth. After fulgurating about one minute it occurred to me that the treatment, which was quite painful, might be having some effect on the peristalsis of the ureters. Observing the orifices, I found to my surprise that both were wide open, golf hole in appearance, with no sign of peristalsis whatever and no indigo-carmine being excreted. Observation was continued for thirty minutes, but the dye did not reappear. A floccule of pus was seen to enter and disappear up the lumen of the right ureter. The total quantity of urine excreted the first six hours following instrumentation was one ounce, and for the next twenty-four hours was only eight ounces, though he was made to drink large quantities of water. For two days he complained of severe aching in his back and legs. Though he had no chill, his temperature was elevated to 101°, he was nauseated and vomited twice. One week later he was feeling as well as ever.

Three subsequent fulgurations were required to destroy the growth. Indigo-carmine was given prior to each of these and its excretion studied as before. Every time there was immediate cessation of urinary flow from the kidneys the anuria persisting from three to six hours. The febrile reaction did not recur.

Case 2.—B. J., a colored man, age 68, was referred because of a suprapubic fistula and inability to void per urethra. Prostatectomy had been performed elsewhere three months previously and he had not been able to void since the operation. On rectal examination no excessive prostatic tissue could be felt. Flexible catheters and bougies could not be made to enter the bladder and I decided to try retrograde catherization by means of a cystoscope introduced through the suprapubic fistula. Indigo-carmine was given in order that the ureteral orifices could readily be seen. The appearance time of the dye was four minutes on the left side and six minutes on the right side. The spurts were strong, the color deep blue, the rate three per minute. The prostatic bed was smooth, the vesical neck entirely closed and covered with mucous membrane completely obliterating the urethral opening. A sound was introduced into the urethra and considerable force used in attempting to reach the bladder, but without success. It was noted that excretion of indigo-carmine ceased rather abruptly soon after rough urethral manipulation began. The lips of the ureteral meati assumed an appearance similar to those in *Case 1.* In 20 minutes the dye reappeared, but not in spurts. It leaked out in a slow, continuous stream, as if there were no peristaltic force behind it. The patient experienced no chill or febrile reaction, but his total urinary output for the next twelve hours was only four ounces.

To a lesser degree this phenomenon has been observed in two other cases in whom various regions of the posterior urethra were stimulated to a point just short of producing actual trauma. Obviously one's opportunities for experiments of this nature are limited by a consideration for the welfare of the patient. But the comparative ease with which some modification of the urinary flow from the kidneys could be produced by posterior urethral stimulation indicates that it is neither rare nor difficult to obtain.

COMMENT

Just how an apparent ileus of the ureteral muscles plus temporary anuria is brought about by this experiment I am unable to explain. Keyser has noted that the ureter is subject to various neuro-motor dysfunctions, especially in women. Reverse peristalsis has been considered rare, though Elliott succeeded in producing it by electric stimulation of the hypogastric nerves, and Cabot and Crabtree observed with the fluoroscope a stone pass from the kidney pelvis into the ureter and rapidly back again. Since fibers from the hypogastric nerves are found both in the posterior urethra and ureters, it is probable that they supply a part of the pathway for a reflex inhibition of both renal and ureteral function.

Such cases bring up the interesting speculation that perhaps this temporary anuria, lasting from a few minutes to a few hours, is not an unusual occurrence in every-day life, and might be caused by various things, such as exposure to cold, excessive sexual indulgence, menstruation, constipation, and pelvic disease. Temporary anuria, plus an inhibition of ureteral peristalsis with failure of the normally impregnable ureterovesical valve would easily allow infective organisms to travel from the bladder to the kidney along the lumen of the ureter and there initiate a pyelitis.

CONCLUSIONS

Though the doctrine of the hematogenous route of invasion in pyelitis is well grounded and widely accepted, it is by no means beyond criticism. The importance of ascending infection may be underestimated.

Two cases of ureteral regurgitation observed by means of the cystoscope and caused by traumatizing the posterior urethra are reported. It is felt that there may be a definite connection between this phenomenon and the disease pyelitis.

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DISCUSSION (*Abstract*)

Dr. R. E. Van Duzen, Dallas, Texas.—I agree with Dr. Finney that the hematogenous route of infection in pyelitis is the most common. Only a small proportion of cases have cleared up after treating pelvic infections and infections of the bowel, while removal of distant infections such as teeth and tonsils has aided in the cure of many of my cases.

We see infections ascending from the bladder into the kidney in senile hypertrophy of the prostate, vesical malignancy and contracted bladders, in which there is ureteral dilatation and incompetency of the ureteral vesical valve. O'Connor produced vesico-renal reflux in rabbits by stimulating the bladder with electric current. He believes that in humans if the peristaltic wave of the ureter arrives at the bladder during contraction of the bladder musculature, reflux is possible, but that this is accidental and plays only a small role. Kretschmer has reported universal dilatation of the ureter and bladder in a case without urethral obstruction, probably due to nerve lesion. In these cases we can expect ascension of the infection from the bladder into the kidney, but in the routine case it plays little, if any, part. Dr. Finney's observations are novel, but I believe the anuria was not due to the reflux, but a reflex phenomenon due to pain. We see this frequently in hysterical people.

Dr. George R. Livermore, Memphis, Tenn.—We know that the mere passage of a ureteral sound often gives rise to fever, chill, and all the evidences of sepsis, but I have never known why. The fact that Dr. Finney has seen the relaxation occur and the reflux follow throws light on this subject.

THE STUDY OF ONE HUNDRED DEATHS FROM DIPHTHERIA IN SOUTH CAROLINA DURING THE YEARS 1926 AND 1927*

By JAMES A. HAYNE, M.D.,
Columbia, S. C.

In May, 1927, an article by Walter W. Lee, M.B., in the monthly bulletin of the Indiana State Board of Health gave an analysis of diphtheria mortality in Indiana during the six months from September, 1926, to February, 1927, and this article contained such interesting data that the State Health Officer determined to make a similar analysis of one hundred deaths in South Carolina.

We have felt for some time that the effort being made through the State Board of Health to reduce the mortality from diphtheria in South Carolina was not proving as effective as it should. When Behring and Roux published their discoveries of the curative properties of diphtheria antitoxin the medical world rejoiced that a discovery had been made that put an end to the fearful mortality of this disease, which is one of the most dreaded diseases of childhood. It has been known as the "strangler" because it chokes children to death. Although antitoxin quickly brought the mortality from diphtheria from 40 in 100 cases to 5, yet this 5 per cent of deaths still continues, and in fact the death rate has slowly risen. The new discovery of the Schick test and the immunization by toxin-antitoxin have gone far toward solving the problem of reducing this hitherto irreducible minimum death rate. South Carolina was the first state in the Union to furnish diphtheria antitoxin free to all its citizens, and after eighteen years of experience with it we feel that the medical profession and the public should be educated as to its value. South Carolina is expending annually about $22,000.00 for diphtheria antitoxin, but if a greater number of children were immunized with diphtheria toxin-antitoxin it would be cheaper and would prevent many cases.

The first table which I submit for the consideration of the medical profession is as follows:

Children under 1 year	16
Children from 1 to 5 years	72
Children from 6 to 10 years	8
Children over 10 years	4

There were of the 100 deaths analyzed, 78 white and 22 colored.

Analysis of these figures will show, first, that contrary to the opinion of some medical writers, negroes are quite susceptible to diphtheria, although not so susceptible apparently as the whites. The next outstanding fact is that from birth to five years of age occurred 88 deaths out of the 100. This means that our preventive measures must be instituted before the school age; for from 6 to 10 years of age, inclusive, there were 8 deaths, and for over ten years there were only 4 deaths. The difficulty of instituting preventive measures in the pre-school group of children can readily be appreciated. The children in school are in groups and can be immunized against smallpox, diphtheria and scarlet fever, but when they are in their individual homes we find it extremely difficult to get them together for administering these preventive measures. However, figures show that if we wish to do anything toward preventing diphtheria we must start in at least in the first years of the child's life, mortality being greatest between 1 and 3 years of age.

The next figures which we wish to call to your attention are:

*Read in Section on Public Health, Southern Medical Association, Twenty-Second Annual Meeting, Asheville, North Carolina, November 12-15, 1928.

Deaths where physician was called on first day	7
Deaths where physician was called on second day	15
Deaths where physician was called on third day	21
Deaths where physician was called within 3 days (The sum of the items above).	43
Deaths where physician was called after third day	57

In other words, 57 of the 100 children who died were seen after they had been sick seventy-two hours. This shows us that a campaign of education must be conducted among the mothers to teach them to recognize readily the first symptoms of diphtheria in their children and to emphasize the fact that, if seen by the physician within the first twenty-four hours few die; if seen within forty-eight hours double that number die, and if calling the physician is put off until after the third day, eight times as many die, in spite of the administration of diphtheria antitoxin.

Analysis of the number of children receiving antitoxin shows as follows:

Ninety-four children received antitoxin of the 100 that died; 42 of them received it in one dose; 48 received it in repeated doses; there was no dosage data in regard to four. Six children did not receive it at all and six did not receive it on the physician's first call. Eighty-one children received 10,000 units or more. There were 31 of these children who were moribund when the physician was called and 38 died within one day after the physician saw them. The children received the following amounts of diphtheria antitoxin:

No diphtheria antitoxin	6
3,000 units	1
5,000 "	3
10,000 "	13
15,000 "	1
20,000 "	30
25,000 "	1
30,000 "	5
35,000 "	2
40,000 "	12
50,000 "	3
60,000 "	10
65,000 "	1
70,000 "	2
80,000 "	2
90,000 "	1
100,000 "	2
Amounts not stated	5

It would seem from this that the children received a sufficient amount of diphtheria antitoxin, as 76 of them received 20,000 units or more. The initial doses given were:

3,000 units	1
5,000 "	5
10,000 "	29
20,000 "	44
25,000 "	1
30,000 "	1
40,000 "	5
Not stated	8
Not given	6

Two reports state that 60,000 or 65,000 units were given, but do not say whether or not this was done in one dose. This would again indicate that 80 per cent received an initial dose of 100,000 units or more and that 51 per cent received an initial dose of 20,000 units or more.

It may be interesting to give the types of diphtheria as stated on the questionnaires sent out:

Laryngeal	56
Pharyngeal	13
Laryngeal and pharyngeal	3
Laryngeal and tonsillar	1
Pharyngeal and tonsillar	2
Tonsillar	3
Laryngeal, pharyngeal and tonsillar	1
Tonsillar and nasal	2
Tonsillar and uvular	2
No clinical signs (ears)	2
Membranous croup	3
Croupous	1
Throat	1
Malignant	1
Back of throat	1
Very severe membrane	1
Naso-pharyngeal	1
Not stated	6

This would indicate that the laryngeal type caused over 61 per cent of the deaths.

The seasonal incidence of the disease as shown by the deaths is as follows:

January 14, February 4, March 5, April 2, May 2, June 3, July 1, August 3, September 14, October 25, November 16, December 11.

This clearly indicates what we have found (long known to be the case) in sixteen years' experience with diphtheria in South Carolina, namely: that the disease commences to climb the middle of August, reaches its height somewhere about the middle of November, and then declines, July being usually the month in which there are fewer cases than at any other time. In a previous paper we have attributed this not to the opening of schools, as it occurs before the opening of schools, but to the dryness which usually prevails in August, September and October, the dust irritating the throat, lowering cell resistance and thus making it a suitable soil for the growth of the diphtheria bacillus.

Dr. W. H. Park of New York, an outstanding authority on diphtheria, brings out the following points in his book, "Pathogenic Micro-organisms":

There is still some difference of opinion as to the proper dosage of antitoxin and as to the method of administration. The amount of toxin in any case of diphtheria is comparatively small. One hundred units of antitoxin would be sufficient to neutralize fifty times the amount of toxin that would kill a six year old child and would render harmless all the toxin present in the most malignant case of diphtheria if the antitoxin could get to the toxin quickly enough. Much more than this has to be given, however, on account of the time required for the antitoxin to reach the

toxin. The antitoxin can reach the toxin only by being absorbed or injected into the blood and then passing through the capillary walls to the tissue fluids and cells. The more antitoxin there is in the blood the more quickly a sufficient amount will pass to the tissues.

How the antitoxin is administered is naturally of great importance. When given subcutaneously, it takes twenty-four hours for the greater part of the antitoxin to be absorbed by the blood. When given intramuscularly it takes about twelve hours. In either case it takes two or three days for its total absorption. When given intravenously, antitoxin passes out to the tissues in two or three hours, that is, ten times more rapidly than by the subcutaneous route, and four times more rapidly than by the intramuscular route. If it were not for the fact that the intravenous method is more difficult to use and that it is more likely to cause serum reactions, this method would be the only one used.

Another important point is the use of single or multiple doses of diphtheria antitoxin. It is very important to remember that antitoxin has no effect whatever on injury that has already taken place and is of no service if the toxin is already united with the cells. The important thing, then, is to give the first dose early enough and large enough, on the first day, if possible. Holding back part of the dose until later delays its action until it can have little or no effect. When the first dose is large enough, and it ought always to be large enough, other doses, although harmless, are absolutely useless. The following single doses for children are advised by Dr. Park:

Mild cases—2,000 to 4,000 units subcutaneously or intramuscularly.

Moderate cases—3,000 to 10,000 units intramuscularly or subcutaneously.

Severe cases—5,000 to 15,000 units intramuscularly or one-half intravenously and one-half intramuscularly or subcutaneously.

Malignant cases—10,000 to 20,000 units, one-half intravenously and one-half intramuscularly or subcutaneously.

Dr. Park recently stated that in many apparently moribund cases anti-streptococcus serum, if administered along with the diphtheria antitoxin, will prevent death. Some of these cases are due to a combined diphtheria and streptococcus infection. Others are due entirely to streptococcus infection and, although swabs may show Klebs-Loeffler bacilli present, the patient may simply be a carrier, not suffering from diphtheria but only from streptococcus infection. This anti-streptococcus serum may be obtained from any biological house.

The facts as we view them that have been brought out by the analysis of these 100 deaths are:

(1) That antitoxin is usually administered in South Carolina to all cases of diphtheria that are either suspected or diagnosed.

(2) That the death rate from this disease is about at a standstill. We get no further decrease in the death rate from year to year.

(3) That toxin-antitoxin must be used to reduce the present death rate and that it must be administered before the child has reached school age, preferably to children between one and three years of age.

(4) That a campaign of education must be carried out to make the mothers recognize that any sore throat in a child should have the attention of a physician and that delay is extremely fatal.

(5) That large doses of diphtheria antitoxin do not prevent the death of children after 72 hours of the disease.

(6) That diphtheria antitoxin to be effective must be administered in an initial dose of at least 10,000 units, or 20,000 units if the case is malignant, and that repeated doses do not seem to have any effect upon the disease.

I wish to thank the physicians who have contributed to the answers of the questionnaires upon which this paper is based, and express the hope that the facts ascertained may be of some benefit.

I have here a chart, prepared by our Vaccine and Antitoxin Supply Depot, covering the diphtheria morbidity and mortality in South Carolina from 1915 to 1928. I ask you to follow me closely in the discussion of this chart as, to my mind, it conclusively establishes several facts in diphtheria control work. It also discloses several facts worthy of our consideration.

You will note that the red lines indicate cases and that the black lines indicate deaths. All figures, rates and percentages are the official figures of the United States Census Department for the Registration Area, except the number of cases, which were accurately compiled by our Vaccine and Antitoxin Supply Depot.

In 1915, we had 1947 cases, of which 160 were fatal, giving us an average of 8.2 fatal cases. As we were not admitted to the Registration Area until 1916, no rate per 100,000 of population was computed. In 1916 we had 1164 cases with 138 deaths. This gives a fatal case rate of 11.8. That year the rate for the United States Registration Area per 100,000 of population was 14.5; the South Carolina rate was 8.5. In 1917 we had 1512 cases with 158 deaths, giving us a fatal case rate of 10.4. That year the rate for the United States Registration Area was 16.5; the South Carolina rate was 9.6.

In 1918, we had 915 cases with 139 deaths,

giving us a fatal case rate of 15.2. That year the United States Registration Area rate per 100,000 population was 13.8; the South Carolina rate was 8.4. The decrease in the number of cases for the years 1916, 1917 and 1918, I think, can probably be explained by the fact that during these years many of our physicians were in the military service. Consequently many cases were not reported. This theory, I think, is borne out by the fact that the deaths for those three years were apparently the same as in 1915 from a fatal case standpoint.

In 1919, we had 2142 cases with 162 deaths, giving us a fatal case rate of 7.5. The rate for the United States registration area per 10,000 population was 14.7; the South Carolina rate was 9.7.

In 1920, we had 2158 cases with 201 deaths, giving us a fatal case rate of 9.3. The rate for the United States Registration Area per 100,000 population for that year was 15.3; the South Carolina rate was 11.9. You will observe that in 1920 we had 16 more cases than in 1919, but looking at the mortality column we find that with only 16 more cases we had 39 more deaths in 1920 than in 1919.

In 1921, we had 2982 cases with 210 deaths, giving us a fatal case rate of 7.0. The rate for the United States Registration Area per 100,000 population for that year was 17.7; the South Carolina rate was 12.3. Comparing the year 1921 with 1920, we find that we had 824 more cases in 1921, yet our deaths increased only 9.

In the year 1922 we had 2959 cases with 189 deaths, giving us a fatal case rate of 6.4. The rate for the United States Registration Area for that year per 100,000 of population was 14.6; the South Carolina rate was 10.6. In 1923, we had 2223 cases with 156 deaths, giving us a fatal case rate of 7.0. The rate for the United States Registration Area per 100,000 of population for that year was 12.1; the South Carolina rate was 8.9. In 1924, we had 2347 cases with 128 deaths, giving us a fatal case rate of 5.4. The rate for the United States Registration Area per 100,000 of population for that year was 9.4; the South Carolina rate was 7.3. In 1925, we had 2613 cases with 127 deaths, giving us a fatal case rate of 4.8. The rate for the United States Registration Area per 100,000 of population for that year was 7.8; the South Carolina rate was 7.0.

Here again you will notice that in 1925 we had 266 more cases than in 1924; nevertheless, there were only 127 deaths in 1925 as against 128 in 1924. In 1926, we had 3001 cases with 152 deaths, giving us a fatal case rate of 5.0. The rate for the United States Registration Area per 100,000 of population for that year was 7.5; the South Carolina rate was 8.3. Here you will notice two outstanding facts. More cases of diphtheria were reported in 1926 than in any prior year. In the whole twelve-year period this is the only year in which South Carolina's rate per 100,000 of population was above the United States Registration Area rate, 7.5 as against 8.3. In 1927, we had 2904 cases with 151 deaths, giving us a fatal case rate of 5.2. The United States Census Bureau at Washington advised me by telegram that the rate for the United States Registration Area per 100,000 of population for the year 1927 was not yet available. The South Carolina rate for 1927 per 100,000 of population is 8.2.

To my mind, in so far as South Carolina is concerned at least, under our present system of a free and unlimited supply of diphtheria antitoxin to all, whether rich or poor, black or white, these figures completely establish the fact that from a mortality standpoint diphtheria has surrendered to science and is under control. The fatal case rate of 8.2 in 1915 rose to 15.2 in 1918, while our physicians were engaged in the military service of this Country. Upon their return in 1919, it promptly dropped back to a rate of 7.5, and while the progress has been slow and year by year the number of cases are constantly on the increase, the rate has fallen until 1927 shows a fatal case rate of 5.2. Covering a period of thirteen years, our morbidity rose to 3001, yet our mortality has never exceeded 210. I ask myself this question, "If we can reduce our fatal case rate from 15.2 to 4.8, what reason can we assign why the mortality cannot be further reduced?" I hope that some of you gentlemen may be able to throw some light on this condition.

From these calculations it is quite apparent that the diphtheria morbidity can neither be checked nor controlled through the administration of diphtheria antitoxin, save perhaps for the very slight reduction by immunizing direct contacts with the immunizing dose of diphtheria antitoxin. If we are to reduce the cases of diphtheria which appear year after year in increasing numbers, we must convince the lay public of the protection to be derived through the immunization of pre-school children with diphtheria antitoxin. All public health officials and the medical profession as a whole must convince the general public of the protection to

be derived from immunization, and that toxin-antitoxin, whether horse, sheep or goat, is not harmful to the child. When the general public gains a realization of the fact that more than a million of children in the United States have been successfully immunized against diphtheria without accident or mishap except in a very few incidents, that diphtheria has been completely exterminated in several cities for more than a year where all of the children have been immunized, that various schools throughout the United States have reported not a single case of diphtheria in a school year where the children have been immunized against diphtheria, then, and not until then, will the value of diphtheria toxin-antitoxin be fully appreciated. It will be appreciated not only from a medical standpoint in safeguarding the life of the child, but a great economic loss will be averted, and the saving on the diphtheria antitoxin bill of this Country will more than offset the amount expended for diphtheria toxin-antitoxin.

In South Carolina diphtheria toxin-antitoxin came into general use in 1926, and while sufficient time has not elapsed to gather and compile any great amount of data, we confidently expect to see a marked reduction in our cases of diphtheria during the year 1929. A slight reduction has already been noted in 1928.

South Carolina was the first State in the United States to recognize the great value of diphtheria antitoxin. In 1910 the South Carolina State Board of Health applied to the State Legislature for an appropriation with which to purchase diphtheria antitoxin and distribute it absolutely free to all citizens of the State regardless of color or financial standing. In South Carolina we believe that the diphtheria carrier is a menace to public health, whether the child be rich or poor, black or white. As the antitoxin is paid for by taxation, to us it seems only fair and just where the taxpayer supplies diphtheria antitoxin to the poor free of charge he should also be entitled to obtain diphtheria antitoxin free of charge for his own use. During our eighteen years of experience in the free distribution of diphtheria antitoxin we have learned much; we have made many changes and modifications in our system of distribution; we feel today that we have perfected a system, not entirely free from defects and abuse, but one which is efficient and economical, and we think that our system of distribution has played a very important part in keeping our diphtheria death rate below the death rate of the United States Registration Area by reason of the fact that diphtheria antitoxin is readily available in every town of 500 population in South Carolina, without any waits or delays and without the signing of any pauper certificates or any red tape of any description.

METHOD OF DISTRIBUTION OF DIPHTHERIA ANTITOXIN IN SOUTH CAROLINA

Many states have written to ask us how we distribute diphtheria antitoxin free to all in South Carolina. At the risk of imposing upon your time and for the benefit of those who are interested in the subject, I shall briefly outline our system of distribution. Years ago we realized that the primary object of the State Board of Health was to prevent and control contagious disease. The State Board of Health, while it should be economical at all times, is not primarily a department of the Government whose principal function is to reduce State appropriations. The time has passed when the biological requirements of a state can be looked upon as a commercial football to be tossed around among competing biological laboratories. There are many factors which enter into the efficient and economical supply of biological products, and it is a great mistake in my opinion for a state to shift about from one commercial laboratory to another for no reason whatsoever except a price concession of a few cents per package. South Carolina followed this procedure until 1920, going from one to another, being influenced largely by price. See what the chart shows from 1915 to 1920. The fatal case rate went up and down, we were in constant confusion, waste was enormous, the service was unsatisfactory, the public complained, physicians complained, we complained, the commercial laboratory complained, and no one was satisfied. In 1921 we realized that diphtheria antitoxin alone would not control diphtheria, but that diphtheria antitoxin handled in a systematic, efficient manner, immediately available at all times to physicians in all parts of the State, even to the smallest towns, and within fifteen minutes' ride by automobile, would control diphtheria.

I would suggest to those who are interested in this part of the campaign against diphtheria to do as we have done in South Carolina, that is, select a commercial laboratory in which you have confidence, give it a general outline of what you want to do and allow it to work out the details of a state-wide distribution system. The laboratories have experience along this line. A plan which would be satisfactory in one state

might not meet the requirements of an adjoining state, but a plan can be perfected which will adequately meet your requirements. Once you have your plan perfected and diphtheria under control, do not make the fatal mistake of abandoning the whole system simply because some corporation offers you a trivial concession in price. Our experience has been that the saving falls far short of compensation for the general confusion and dissatisfaction involved in substituting one system for another, and is not worth while.

In South Carolina we maintain and operate what we term our Vaccine and Antitoxin Supply Depot under a trained personnel, and all expenses in connection with this depot are defrayed by the commercial laboratory. The personnel of this Depot is under our direction and of our selection. All of the details in connection with the handling of biological products under the State Board of Health, such as bookkeeping, shipping and compiling of statistics, are attended to by this Depot. The State Board of Health is completely relieved of this work. All that we have to do is to audit the account when it is rendered once each month. This Depot has refrigerating facilities and the entire system throughout the State is supplied from it. Under the supervision of the Depot there are some 200 distributors, at least one in each town of 500 population. Each distributor is supplied with requisition blanks in triplicate. These blanks give the name of the attending physician, the name, age, sex and color of the patient, together with the amount of antitoxin used. It is signed by the physician who requisitions it and the distributor certifies that the antitoxin called for by the requisition was delivered to the physician in proper condition. Each distributor receives a commission on all antitoxin distributed through his station, which commission does not become payable until his requisitions are on file in the State Board of Health office. The diphtheria antitoxin so used does not become a charge against the State until the requisitions are on file at the State Board of Health office.

By this method it will readily appear that the State of South Carolina pays only for the diphtheria antitoxin actually used and certified to by the attending physician and the distributor. Thus all waste is eliminated. Our Antitoxin Supply Depot keeps a record very similar to an individual bank record of checks and deposits. The distributor starts out with an original stock supplied by the Antitoxin Supply Depot. When the distributor sends in a requisition showing that he has distributed 10 packages of 10,000 units each, our Supply Depot immediately deposits 10 packages of 10,000 units with him to replace that drawn out on requisition. The Depot at all times knows what stock each distributor has. During light seasons the stock is reduced; during the winter months, or when epidemics occur, the distributor's stock is increased, by which method diphtheria antitoxin in adequate quantities is always available. Complete records are kept and at the end of the month, each month, the Supply Depot submits to the State Board of Health a statement showing the amount of antitoxin distributed by each distributor, giving the number on each requisition, and from these requisitions we are enabled definitely and accurately to determine our progress in controlling diphtheria. As was stated, all of our records are kept in triplicate, one set being on file in the office of the State Board of Health, one set in the office of the Antitoxin Supply Depot, and one set being retained by the distributor. This is a public record, subject to inspection and audit by all proper parties. We have followed this procedure now for seven years and, as I said, while it is not absolutely perfect, we feel that it is efficient and economical, and it has never failed to render prompt and adequate service under epidemic conditions.

DISCUSSION (*Abstract*)

Dr. W. A. Mulherin, Augusta, Ga.—The good old state of South Carolina is to be congratulated on being the first state in the Union to dispense, free of charge, diphtheria antitoxin.

Dr. Hayne rightly calls attention to the fact that the administration of toxin-antitoxin is not giving the results that we naturally should expect from its use. The reasons for this, I believe, are: a new crop of babies appears every year, furnishing more fertile soil, and the medical profession is not properly educated as to the importance, and almost complete dependability, of this preventive measure. Of more importance than all is the fact that toxin-antitoxin is not given early enough in the first year of life.

Fully 75 per cent of mortality due to diphtheria will be found in the first five years of life. Hence, early administration of toxin-antitoxin should be stressed. One should not wait till the child is of school age. Until the medical profession is educated to the point of realizing that toxin-antitoxin is protective, and should be given to all babies between the ages of six and nine months, our fight against diphtheria will not be successfully waged.

As regards dosage of antitoxin, I realize that Dr. Park is a very high authority on all matters pertaining to proper treatment of diphtheria, and that, theoretically, it may be true that 5000 to 10,000 units is as large a dose as should be used for neutralizing the poison

generated by diphtheria germ. From my experience gained in twenty-eight years of practice, I would be fearful to limit myself to this dose in treating my cases. My practice has been, and is today, to give 10,000 units in one dose in an ordinary case of diphtheria, and 20,000 units for all cases of laryngeal and nasopharyngeal cases. I believe the old practice of biological houses putting 20,000 units in one dose should be continued, instead of administering two 10,000 units to the child.

Dr. Hayne calls attention to cases of diphtheria that have a mixed infection, say streptococcic or Vincent's organism. When a child does not respond to a full dose of antitoxin administered in the first one to two days of illness, mixed infection should be suspected, and if culture or smear verifies suspicions, then anti-streptococcic serum, or sulpharsphenamine, given intramuscularly, will give most brilliant results and save life.

Dr. E. L. Bishop, Nasheville, Tenn.—I think I can best support Dr. Hayne's thesis by expressing something of our own point of view and discussing the methods of approach in Tennessee.

Dr. Hayne stated that 88 of the 100 deaths were of children under six years of age. In Tennessee 73 per cent of all diphtheria deaths are of children under six years of age and 94 per cent of all deaths are of children under ten years of age. It is obvious that Dr. Hayne is sound in stressing the importance of immunization of children under six years of age, for if we were to immunize every susceptible child from six years of age through the school age we could prevent only about one-fourth of the total deaths, whereas on the other hand dealing with a much smaller group it is possible to prevent three-fourths of the total deaths. Much of the activity in the immunization of children against diphtheria has had comparatively small value since it was directed to the school age rather than the pre-school age group.

In Tennessee, believing that we can control diphtheria only by the direction of our activity to the first few years of life, we have imposed the definite restriction that toxin-antitoxin will be given in our immunization clinics only to children under ten years of age. In a few instances the field staff has to accept children over ten, but these constitute a very small fraction of the total immunizations. We have in this way limited our problem very materially since there are only about 300,000 children in the State who are included in the first five year age group of life. If we extend the period to the first ten years, we find that about 600,000 of our total two and one-half millions population is involved. Subtracting from this the fraction of the population that will be immunized through full-time county health departments and through city health departments, it is probable that our total problem in connection with unorganized counties will deal with less than 200,000 immunizations. The State is attempting direct service only in counties which have no full-time local health work and is doing its work in cooperation with county medical societies. Toxin-antitoxin is supplied for the use of physicians, or field workers are put into the county to work with the physician in the administration of toxin-antitoxin. This work began in August and already 16 of the 72 unorganized counties have been reached. Another point which we observed in the selection of counties is that priority is given to counties which have shown the highest mean death rate for the past ten years. Our analysis shows that twenty-four of our counties have a mean ten-year rate above fifteen, while seventeen others range from 12 to 14. Thus our heaviest mortality is found in forty-one of our total ninety-four counties. I believe that we should in formulating a program for administration of toxin-antitoxin base our activity on a study of the age and geographic distribution of the disease, for if we do so we can either control or reduce the incidence materially with a minimum outlay of funds. We confidently expect this result in Tennessee on the completion of our present activity.

Col. Charles R. Keiley, Richmond, Va.—Our health department does not distribute antitoxin freely. In fact we demark so far as possible preventive work and remedial work. We devote our energies exclusively to prevention. Last year we had 156 deaths from diphtheria.

Last year there unexpectedly fell into our lap, by reason of a change in the financial or tax system of the State, a windfall of approximately one hundred thousand dollars. Dr. Williams decided to use the bulk of it in an effort to lower the diphtheria death rate. We definitely knew that we could lower the rate if we could evolve a workable plan for the wholesale giving of toxin-antitoxin.

We immunized within twelve months several thousand more school children than there are in South Carolina and, in face of a nation-wide increase in the incidence and mortality of diphtheria, we lowered our sickness rate nearly 35 per cent and our death total from 228 to 156, by far the lowest record in our history. Thanks to that beneficent institution which Dr. Hayne mentioned, the U. S. Bureau of the Census, we are able to send out in the same franked envelope which carries a birth certificate, a little slip which reads: "Give your child toxin-antitoxin when he is six months old." A result of this message is that quite an appreciable number of very young children are immunized by their family physicians.

In Virginia we have a statute known as "The West Law." It is a plan of cooperation by which our educational system and our public health system are coordinated for the purpose of promoting the physical welfare of school children; and, through the operation of this law we get the parents to permit their children in school to be protected and to bring their younger children to our clinics for protective treatment.

We had toxin-antitoxin clinics all over Virginia, and we frequently found that our totals exceeded the entire school population of the county. Of course, the explanation was that we had secured a large number of pre-school children. We found that mothers were bringing their babies for great distances over mountains to have them protected. In a tidewater county one patriarch came with seventeen children in his wagon. We have no difficulty in getting attendance. Our only trouble is financial.

This year we are putting on a campaign in the cities of Virginia, and it is wonderful to witness the zeal with which the city health officers are tackling the job. Virtually every city health officer in the State attended the conference we called for the purpose of discussing the year's work, and I believe that I am safe in saying that the campaign will be worked aggressively in every city of Virginia.

Dr. Paul Eaton, Augusta, Ga.—Much more frequently than is suspected scarlet fever and diphtheria are associated. I have charge of the public health laboratory in a town of 60,000 population and twice the last year I have had occasion, accidentally, to prove that this is the case.

In each case there was a suspicion of scarlet fever, but the physician was wise enough to take a culture from the throat and at the same time to administer scarlet fever anti-serum. In each case the fever was cleared up in twenty-four hours, the rash disappeared and the diphtheria developed, to be cured by a further administration of antitoxin.

Dr. C. L. Outland, Richmond, Va.—In malignant diphtheria I notice that one of the German authorities has stressed the point that there is usually a streptococcic infection along with the diphtheria. Very often malignant diphtheria is nothing more than an acute streptococcic infection plus the diphtheria, which may be of mild form.

If ten to twenty thousand units of antitoxin is enough for an ordinary case, I think it very important not to continue giving antitoxin when after a reasonable length of time results have not been attained. In the meantime, to give 20 to 50 c. c. of polyvalent streptococcic antitoxin will be a better treatment.

Dr. A. T. McCormack, Louisville, Ky.—We have been able to immunize about 75 per cent of the school population and 50 per cent of pre-school children with toxin-antitoxin. We have not gotten over the entire area, but we have reduced the number of those conveying the disease so considerably that the morbidity and mortality in the flood counties have almost disappeared and the incidence of diphtheria is as low as that of smallpox.

I was particularly gratified to hear Dr. Hayne's statement in regard to the dosage of antitoxin. I think we have been administering an unnecessary dose; that is, too large, in many cases. If the case is sufficiently severe, 10,000 units, given intravenously, are sufficient, and we have adopted a plan in that respect. Under our Bureau of Venereal Diseases we have a clinic in all counties now except eleven, conducted by county medical societies. So, one physician has been trained in intravenous medication. It requires an especially expert technician to give antitoxin to children, and we found this to be a very distinct need in the administration of antitoxin.

We have cut the death rate a little more than 55 per cent over the previous ten-year period.

In the negro population the death rate has been reduced to 68 per cent by the same procedure.

THE SPECIFIC ACTION OF BLOOD TRANSFUSION IN THE HEMOLYTIC ANEMIA OF PREGNANCY*

By WILLIAM ALLAN, M.D.,
Charlotte, N. C.

The hemolytic anemia of pregnancy occurs chiefly in women under 30, and runs an acute course without remissions. Starting in the latter half of pregnancy, it is generally discovered soon after delivery, which is apt to be premature. Because of the presence of fever which is sometimes accompanied by leucocytosis,[1] a diagnosis of puerperal sepsis is often made and symptomatic treatment instituted. This expectant plan of treatment results in a mortality rate[2] varying from 35 to 80 per cent. As contrasted with this experience, a review of the literature shows that blood transfusion after delivery can reduce this mortality to less than 10 per cent.

The appearance of the patient, her symptoms and the clinical and post-mortem findings parallel rather closely those of chronic pernicious anemia and need not again[2] be reviewed in detail.

The cause of this anemia is thought by some to be a toxic agent derived from the pregnant uterus,[5 16] since the anemia occurs only during pregnancy and frequently disappears spontaneously and permanently soon after delivery. However, in some cases the condition grows progressively worse for some weeks or months after delivery and normal involution of the uterus, so that some other factor must be invoked to explain its continuation.

By many this anemia is thought to be due to sepsis and in some cases it is undoubtedly intensified by complicating infections, as illustrated by Larribere's case[17] of malaria and pregnancy and by the cases cited below, but in many instances there are no signs of infection.

Case 1.—A white woman, age 16, was seen at Mercy Hospital with Drs. Martin and Wannamaker. She had been admitted March 3, four and a half months pregnant, and operated upon for appendicitis.

March 7 her temperature rose to 104° and she continued to show a high septic type of fever with an average leucocyte count of 15,000 until after transfusion ten days later. Ureteral catherization March 11 and 15 was reported negative and the patient aborted March 12.

When I saw the patient, March 16, anemia was the most striking symptom. Blood examination showed hemoglobin 35 per cent, red count 1,840,000, color

*Read in Section on Medicine, Southern Medical Association, Twenty-Second Annual Meeting, Asheville, North Carolina, November 12-15, 1928.

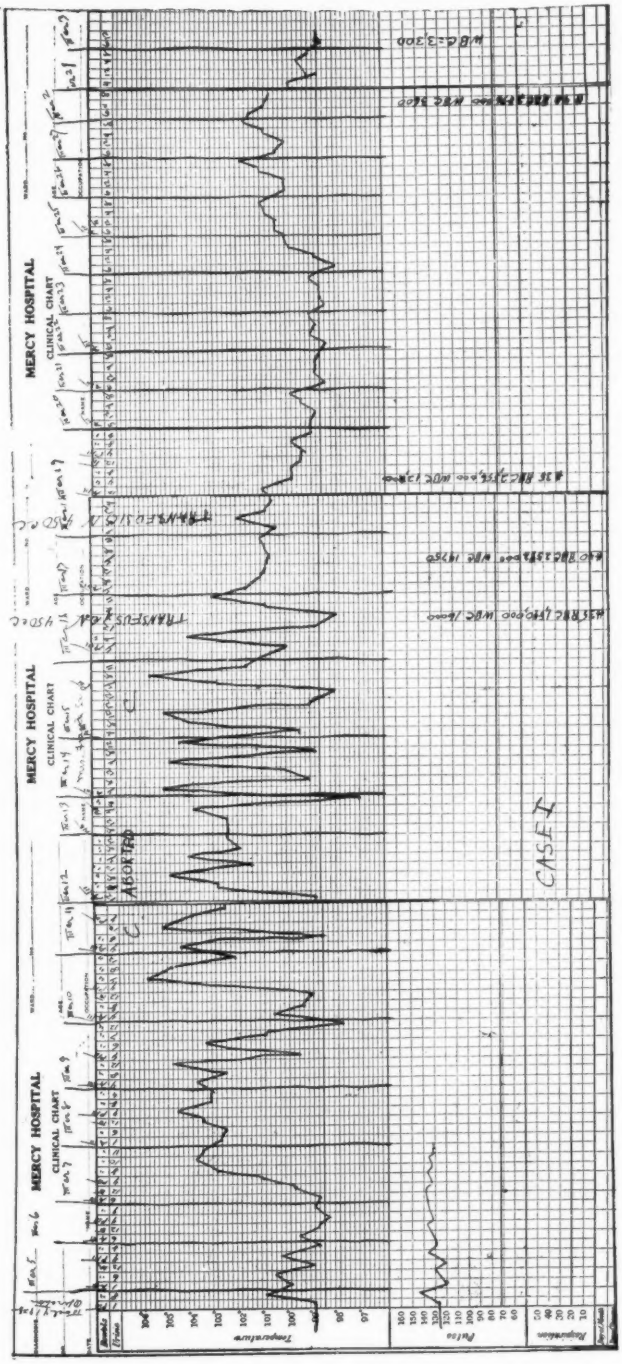


Fig. 1. Case 1

index 1, with 16,000 leucocytes, 88 per cent of which were polymorphonuclears. After two blood transfusions of 450 c. c. each March 16 and 18, the temperature and leucocyte count promptly returned to normal March 19. The next week the patient had fever of 100 to 101° for four days with hemoglobin 40 per cent, red count 2,896,000, but with only 3,500 leucocytes on two consecutive days. She was discharged from the hospital on March 30 with normal temperature. Two weeks later she had some fever again with definite pyelitis, which was easily controlled by lavage of the kidney pelves. At this time, April 14, the blood showed hemoglobin 65 per cent, red cells 3,400,000, with a leucocytosis of 17,000. Blood transfusion in this case corrected the anemia without abolishing the infection.

Case 2.—A white woman, age 22, was seen September 28, 1928, with Dr. R. W. Petrie at the Lenoir Hospital. She had a daily rise of fever reaching 100 to 101° and was strikingly pale. She said she had been pale all her life, but was thought to have been paler than usual during pregnancy. However, she had felt well and worked hard until labor began, August 28, at full term. Labor was normal, with no unusual bleeding. The puerperium was normal until fourteen days after delivery, when pleurisy developed over the upper half of the right chest, and a week later milk leg developed on the left side. She was admitted to the hospital September 22, with hemoglobin less than 20 per cent. When I saw her, September 28, the hemoglobin was 35 per cent, red cells 1,848,000, color index 1, leucocytes 14,400, polymorphonuclears 68 per cent, lymphocytes 30 per cent, eosinophils 1 per cent, basophils 1 per cent. There was marked irregularity in size and shape of the red cells and a number of normoblasts were seen. Transfusion in this case had been attempted on admission, but failed because of technical difficulties. The patient was unable to take liver, but her family physician, Dr. C. R. Russell, of Granit Falls, N. C., put her on liver extract with iron and arsenic and October 27 reported that she was recovering rapidly.

	Hem.	R. B. C.	C. I.	W. B. C.	Trans.	Result
Smith (3)	30	1,080,000	1.2	3,800	1	Recovered
	40	1,300,000	1.	2,600	1	Died (pneumonia)
	10	570,000	1.	4,000	1	Recovered
	20	1,280,000	0.95	3,800	1	Recovered
	15	970,000	0.9	5,800	1	Died (too little)
Larrabee (4)	12	590,000	1.	3,100	1	Recovered
	29	1,420,000	0.97	2,000	1	Recovered
	19	660,000	1.4	6,400	4	Recovered
	28	1,080,000	1.2	2,500	2	Recovered
Roland (5)	25	600,000	2.4	3,200	3	Recovered
Gallupe & O'Hara (6)	35	1,790,000	1.	4,300	1	Recovered
	30	2,000,000	0.9	7,500	1	Recovered
Schmidt (1)	24	1,280,000	1.	2,800	2	Recovered
	10	800,000	0.62	2,400	1	Recovered
	23	1,800,000	0.7	4,900	2	Recovered
Bonta (7)	17	1,000,000	0.85	low	4	Recovered
	10	780,000	0.65	7,000	5	Recovered
Murdock (8)	40	1,660,000	1.2	6,600	1	Recovered
	33	1,600,000	1.	5,200	2	Recovered
Hoskins (9)	28	1,870,000	0.8	2,600	1	Recovered
Heim (10)					1	Died (too late)
Ottingen (11)					1	Recovered
Reist (12)					4	Recovered
Levy-Solal & Tzanck (13)		2,600,000			4	Recovered
		750,000			4	Recovered
Allan (2)	25	832,000	1.5	2,700	2	Recovered
	25	742,000	1.8	6,600	2	Recovered
	10	960,000	0.53	2,500	1	Recovered
	35	1,840,000	1.	16,000	2	Recovered
	35	2,688,000	0.7	4,100	1	Recovered
	35	1,960,000	1.	18,000	1	Recovered
	15	910,000	0.83	78,400	1	Died
Brindeau (14)	16	634,000	1.3	3,080	4	Recovered
Atzerodt (15)	23	1,310,000	0.88	2,700	2	Recovered

Case 3.—A white woman, age 23, was sent to the hospital October 31, 1928, with coma, fever, rapid pulse and respiration and extreme pallor. The family and past history were unimportant. She had gone into labor October 26, at the end of the eighth month, and after four hours was delivered of a blue baby which lived only a few hours. Very little blood was lost. This was her fourth pregnancy in four years. The first three had been normal and resulted in healthy babies. Two days before her last labor and again on the day of delivery the family physician was called to see the patient because of sore throat. The day after delivery the woman seemed bright and ate well. The next day she ate poorly and had backache. The third day she vomited a great deal and was delirious at times. The fourth day she vomited all day and became comatose. The fifth day she was admitted in coma with axillary temperature 103.8°, pulse rate 180 and respiratory rate 60. There were fresh hemorrhages in the retinae, but no hemorrhagic manifestations in the skin, which was extremely pale but not icteric. The throat was nearly white, no pathologic lesions being seen. The lungs were clear. Heart size was normal, heart sounds revealed a systolic murmur all over the precordia; the rate was 180, regular. Blood pressure was 100 systolic and 30 diastolic. The abdomen was flat and relaxed, with the uterus nearly to the umbilicus. Neither liver nor spleen were palpable. There were no enlarged glands. Examination of the blood showed hemoglobin 15 per cent, red cells 910,000, color index 0.83, leucocyte count 78,400, with polymorphonuclears 59 per cent, lymphocytes 35 per cent and myelocytes 6 per cent. The red cells varied greatly in size, shape and staining reactions and both normoblasts and megaloblasts were present. There were 8 per cent of reticulocytes. Fragility tests showed that hemolysis began at 0.48 per cent sodium chlorid solution and was complete at 0.36 per cent. The icterus index was 11.5 and van den Bergh's immediate, direct and delayed reactions were both positive.

The urine showed a good trace of albumin and was at 4:30 p. m. strongly positive for urobilinogen, but otherwise negative. The patient was given a direct blood transfusion of 750 c. c., after which the respiration dropped to 52 and the pulse rate to 148. Two hours later there was a reaction from the transfusion, with flushed face, extreme restlessness, respiration 40, and pulse 150 and irregular. At 9 p. m. the patient was pale, quiet, still in deep coma, with respiration 33, pulse rate 160, with auricular fibrillation and axillary temperature 106°. She died at midnight with axillary temperature 107.4°.

In this case the anemia was complicated by sore throat starting before labor and the possi-

bility of puerperal infection following labor. A similar case[2] admitted in coma, but without any complicating infection, was promptly relieved by blood transfusion. I have seen the same blood picture with coma following a hemolytic crisis in hemolytic icterus.[21]

The similarity of the clinical picture of the pregnancy anemia to that of chronic pernicious anemia is so striking that they have rarely been differentiated, and the response of the pregnancy anemia to liver feeding is apparently so much like the response obtained in pernicious anemia that it naturally suggests a similar etiology.

Means and Richardson[18] consider pernicious anemia a deficiency disease in which adequate liver feeding will supply the specific substance necessary to hold the symptoms in abeyance, but without removing the causative factor. The anemia of pregnancy may prove to be the same sort of deficiency disease, but with this difference, that when once the symptoms are abated complete recovery speedily follows. The anemia of pregnancy differs also in that blood destruction may be a much more important factor than lack of blood formation. Liver feeding in pernicious anemia seems to result chiefly in the resumption of erythrocyte formation; in the pregnancy anemia liver feeding or post-partum blood transfusion has the same effect, as is indicated by the reticulocyte curve in the cases reported below, but in addition seems to stop the increased hemolysis, as indicated by the fall in icterus index with the disappearance of urobilinogen from the urine.

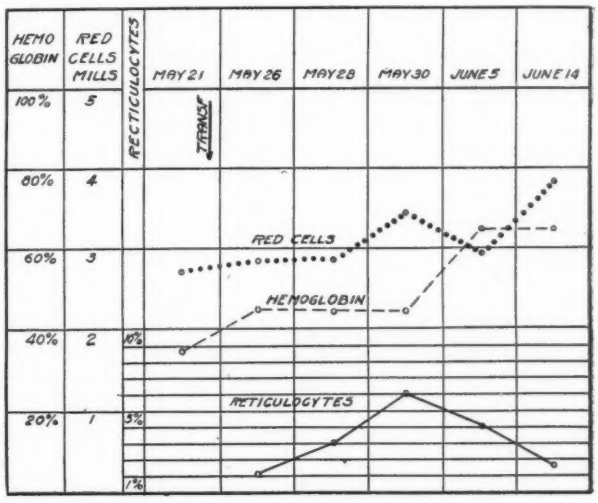
The response of the anemia of pregnancy to protein therapy is interesting. As shown by the table above, blood transfusion after delivery has a specific curative action, but all observers are agreed that blood transfusion before delivery has no such action.[19]

Last year Murdock recommended liver feeding in the mild cases and reported one case in which after delivery liver was fed with improvement. Matters has also reported a successful case of liver feeding after delivery, but Brindeau's patient was about to die after a month of liver feeding, post-partum, when transfusion rescued her.

Much more important, however, are the two cases[17 20] reported in the French literature this year, in which liver feeding before delivery abolished the anemia and enabled these women to go through normal labor at term. Deschamps and Froyez' patient was given a daily ration of 250 grams of liver for twenty-five days during the seventh month of pregnancy, and Larribere's patient was given the same amount from the beginning of the eighth month through the puerperium. Further reports on the response of the anemia of pregnancy to liver feeding both before and after delivery are needed, both to establish its value in this condition and in order that light may be shed on the mystery of chronic pernicious anemia.

Case 4.—A white woman, age 27, was seen May 21, 1928, with Dr. Matthews at the Mercy Hospital, five days after the birth of twin boys. The patient had had one normal labor and pregnancy eight years before. The present pregnancy and labor were normal, although the husband said his wife had looked unusually pale and sallow for the last month. Her temperature was 101.4°, pulse 132 and respirations were 22. The hemoglobin was 35 per cent, red blood count 2,688,000, color index 0.7, and leucocyte count 4,100. Fever had been present since the day after delivery and herpes appeared on the upper lip and right edge of the tongue May 21. Physical examination was negative, except for pallor and a barely palpable spleen.

May 22 she was given 500 c. c. of blood by the citrate method. May 26 the hemoglobin was 45 per cent, red count 2,880,000, white count 12,100, with polymorphonuclears 86 per cent, lymphocytes 11 per cent, and myelocytes 3 per cent. There was considerable variation in size and shape of the red cells,



CASE IV

Fig. 2, Case 4

with both normoblasts and megaloblasts present and reticulocytes 1 per cent.

May 28 the hemoglobin and red count were the same as on May 26, leucocytes 14,000, with polymorphonuclears 78 per cent, lymphocytes 16 per cent, neutrophilic myelocytes 6 per cent, reticulocytes 3 per cent and temperature normal.

May 30 the hemoglobin was 45 per cent, red count 3,424,000, color index 0.66, leucocytes 11,500, with polymorphonuclears 66 per cent, lymphocytes 26 per cent, myelocytes 7 per cent and eosinophils 1 per cent. There was great variation in the size of the red cells; a few normoblasts were present and 6 per cent reticulocytes.

June 5 the hemoglobin was 65 per cent, red count 2,950,000, white count 9,200, there were no normoblasts, and reticulocytes 4 per cent. June 14 the hemoglobin was 65 per cent, red count 3,750,000, white count 7,900, with normal differential count, no normoblasts, and 1.5 per cent reticulocytes.

Case 5.—A white woman, age 36, was seen with Dr. C. S. McLaughlin, October 12, 1928, twelve days after the third labor. The first two labors and pregnancies were normal, fifteen and thirteen years before. Early in the eighth month of this last pregnancy edema appeared, with a rise of blood pressure to 170. The patient was put to bed for two weeks on a restricted diet. Some pallor was noted before labor, which came about three weeks early. When seen she complained of headache, nausea and vomiting, looked very pale and sallow, with pulse 120, respirations 20 and blood pressure 130. There had been fever the ninth and tenth days after delivery. Physical examination showed herpes labialis, eye grounds and sinuses negative, a loud systolic murmur at the apex, and intense anemia. The urine showed urobilinogen. Blood examination showed hemoglobin 35 per cent, red cells 1,960,000, color index 1, great variation in size, shape, and staining reaction of the red cells with 180 normoblasts and 360 megaloblasts per cubic millimeter. There were 18,000 leucocytes, with polymorphonuclears 77 per cent, lymphocytes 18 per cent, eosinophils 3 per cent, basophils 1 per cent, neutrophilic myelocytes 1 per cent, reticulocytes 1 per cent, icterus index 6.8 per cent. Van den Bergh's immediate direct reaction was negative, and delayed reaction positive. The fragility of the red cells in salt solution showed hemolysis beginning at 0.42 per cent, complete at 0.34 per cent. She was given 500 c. c. of blood by direct transfusion.

October 14 the headache, nausea and vomiting had disappeared and the appetite returned. October 17 hemoglobin was 35 per cent, red cells 1,680,000, leucocytes 7,800, with normal differential count, normoblasts were present, and there were 1.5 per cent reticulocytes.

October 22 the hemoglobin was 30 per cent, red cells were 1,456,000, leucocytes 11,000, and reticulocytes 7 per cent. October 24 hemoglobin was 35 per cent, red cells 1,672,000, leucocytes 10,200, reticulocytes 9 per cent, icterus index 7.5, van den Bergh's immediate direct reaction negative and delayed reaction positive. October 29 hemoglobin was 45 per cent, red cells 2,130,000, leucocytes 15,300, polymorphonuclears 80 per cent, lymphocytes 20 per cent, normoblasts present, reticulocytes 8 per cent, and icterus index 6.3.

November 6 the hemoglobin was 55 per cent, red cells were 3,040,000, leucocytes 13,900, no normoblasts, reticulocytes 3 per cent, and icterus index 4, van den Bergh's immediate direct reaction was negative, delayed reaction positive. This picture of blood regeneration after transfusion is like that seen in chronic pernicious anemia after liver feeding.

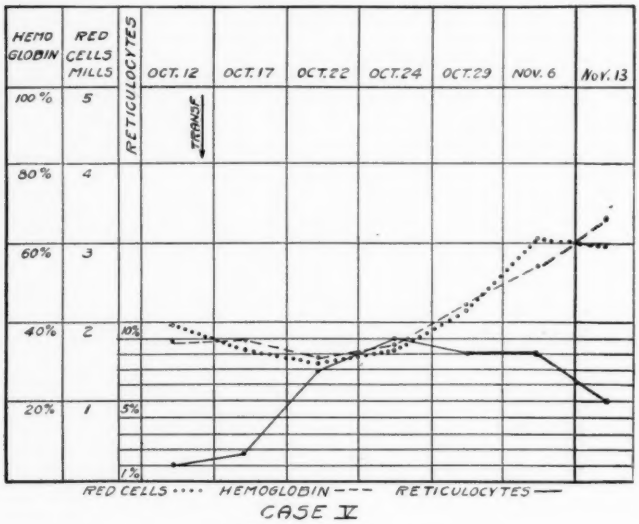


Fig. 3. Case 5

CONCLUSIONS

After delivery blood transfusion has a specific curative action in the hemolytic anemia of pregnancy and should be used promptly in all severe cases.

The reports to date indicate that liver feeding in this anemia has a specific curative action before as well as after delivery. This response to liver suggests that the hemolytic anemia of pregnancy may be a deficiency disease in the same sense in which pernicious anemia has been described as a deficiency disease.

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DISCUSSION (*Abstract*)

Dr. W. R. Houston, Augusta, Ga.—The therapeutic effect of transfusion is so prompt and brilliant that it is comparable to the action of quinin in malaria. I recall having seen about four cases, all of them occurring after delivery. They all recovered, three of them very promptly. After transfusion they showed an almost magical transformation within a day or two. The fever subsided, the subjective conditions began to mend, the restoration of the blood began. The fourth case, when consulting me, was already on the way to recovery. Her case had been diagnosed malignant endocarditis, which obviously was mistaken, and with the help of liver extract she soon regained a normal blood picture.

One of these cases interested me particularly when I saw her again not long ago after a lapse of years. This young woman, with unbridled philoprogenitive recklessness, went on after her first attack to bear three more children, and in none of the other cases of confinement did she have any trouble at all. That made me think that this condition might be due to something closely related to the toxemia of pregnancy. We know that one attack of eclampsia usually confers immunity against future attacks, and it would appear that one attack of this condition conferred immunity against future attacks.

We have come in recent times to think of the reactions of the hematopoietic organs in very general terms. For instance, we think of leucocytosis as being not the only way in which the blood depots respond to stimulus. We may have a leukemic reaction on the one hand; on the other we may find an extremely low white count in the picture of agranulocytosis. We have monocyte reactions as well as polymorphonuclear leucocyte increases. These differences in blood reaction are probably due not so much to the kind of stimulus as to some constitutional factor that gives the type of response in the given case. In the pernicious anemias of pregnancy it may turn out that there is a constitutional factor yet to be defined.

It is very important for it to be known universally that here is a disease which demands recognition, that it is not extremely uncommon, and that we have for it a specific and brilliant cure.

Dr. Allan (closing).—Dr. Houston brought out the question of recurrence. There have been about ten reports of recurrence, although I think Balfour, in India, and I are the only ones who have actually seen individuals through two of these attacks. A woman was sent to me in coma, with very rapid respiration and pulse rate. Transfusion brought about recovery and she went home in a week or two. She started another pregnancy and at the end of the year, about two weeks after delivery, her red blood cells had dropped below a million, and I had to transfuse her again.

Some of the Germans have suggested sterilization to prevent recurrence, but this seems to me absurd. Under proper supervision this anemia of pregnancy can easily be controlled.

VALUE OF CHILD TRAINING IN PREVENTION OF MENTAL DISORDERS*

By NEWDIGATE M. OWENSBY, M.D.,
Atlanta, Ga.

The general practitioner who does not comprehend our nomenclature neither knows nor cares anything about the controversy between structuralism and functionalism, the principles of genetic or comparative psychology, the behavioristic point of view, the Gestalt-Theorie, child psychology, or the average psychobiological, psychopathological or psychogenetic theories taken up by the neuro-psychiatrist. He is interested, however, in any presentation of scientific facts, how they are discovered, and their position in the comprehensive system of scientific knowledge, provided they are presented in such a way that he can understand them. We are aware that all true scientific knowledge must necessarily be obtained by strenuous intellectual effort and intensive research, but in the dissemination of this knowledge we are too prone to forget that the very people who could use it to best advantage are denied the privilege because obtuse technical appellations known only to the initiated are used. I shall disregard insofar as possible the nomenclature of the psychiatrist, and present this paper in the medium more clearly understood by the general practitioner.

It is axiomatic that the seeds of mental ills are most often implanted during infancy or early childhood. However these seeds may have been sown, it is evident that they must be fertilized to produce germination and grow eventually to maturity. Since the family physician comes in contact with a greater group of children in their infancy and early childhood than does any other

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class of physician, it is of urgent necessity that he be informed of the manner in which these seeds are usually permitted to germinate in order that he may set aside certain antiquated notions which have been instilled so deeply into him that his vision has become perverted or else obliterated entirely. The time allotted to this paper is not sufficient to combat all antiquated notions with a lengthy discussion of them, so I shall simply remind the profession of one important point to consider and it is this: the average inmate of an asylum enjoys perhaps better physical health than does his brother or his sister of the same economic and social status on the outside. I shall also like to call attention at the same time to the fact that no authentic textbook on psychiatry in recent years has given masturbation as the main etiological factor in the production of insanity.

In order to make the idea convincing that the seeds of all mental ills are sown during infancy or early childhood, it is necessary to emphasize the fallaciousness of the idea of inheritance that is so deeply imbued in the medical profession. We may come into the world with an anatomically defective brain, or present physical characteristics of our parents, but if the theistic philosophy or Christian religion is correct we are endowed with mind or soul by a Supreme Being, and this is not inherited. Moreover, it does not matter whether we be savage or Christian, this same Being gives each member of the human race a distinct and separate soul or mind. Therefore it is correct to surmise that we do not inherit peculiarities of behavior, personalities, or our psychological outlook, but develop them as a result of our environment, associates and training.

Since the environmental conditions of the savage are entirely different from those of civilized man, he must necessarily receive a different training that he may make his social and everyday working adjustments and contacts; hence, the difference in his customs, manner of worship and psychological outlook. If a savage child were taken from his family at the age of six months and reared by a civilized family he would have the same habits and customs as his foster parents. The same may be said of the child of a Christian were the experiment reversed. Then should this Christian child be returned to his real parents after he had reached adolescence, to civilized surroundings and environments, I strongly suspect that his associates and even his family physician would regard him as mentally abnormal if they were not acquainted with the circumstances of his early training. All this could occur and yet he would be physically and mentally normal for the time being. What his subsequent reactions would be to his changed surroundings would depend entirely upon how strongly his emotional habits had been fixed, and the character of the images impressed upon him during infancy and early childhood, and the restrictions placed upon him. If these were sufficiently great to prevent him from making his adjustments, he would doubtless become morose, introspective, self-analytical, resentful, and eventually he would develop a psychopathological disorder.

We have observed such experiments in wild animals and more particularly in the higher types of the ape family which have been captured and placed in zoos in various parts of the world. Animal trainers state that very few, if any, of the higher types of the ape family survive being taken out of their normal environment, and if they are taken into captivity death soon follows due either to suicidal attempts or as the result of homesickness or melancholia. Again we observe that attempts on the part of explorers and missionaries to remove savages of the adolescent or later ages has always resulted disastrously, yet no anatomical lesion could be found in the brain or bodily organs to account for their mental abnormality. Had either the savage or the ape been permitted to remain among his fellows, in his accustomed environment, it is likely that he would have continued to enjoy good mental and physical health.

With children of the civilized community there are certainly a very great number who are reared in surroundings which prevent their making their everyday working or social adjustments. When they show either mental or physical reactions resulting therefrom the family physician is called and instead of investigating their training he immediately begins a search for a physical cause to serve as the condition responsible for the manifestation. Should his investigation fail to bring forth even a decayed tooth or a hypertrophied tonsil, then, and only then, must some collateral ancestor, no matter how remote, bear the blame, especially should there be no closer relation presenting mental symptoms or conduct disorders.

Should the child be incorrectly taught, or led to infer without teaching directly, that any of the processes of life are disgusting or unrefined, that certain normal instincts are sinful, as has

been noted in most sections of the United States, then indeed will he have a most difficult time in later years in making the adjustments necessary for health and happiness in this very practical world. However, because of the antiquated notions which still persist, he either will be branded as a case of "nerves" and permitted to drift until he eventually reaches a haven in a psychosis, or else have every removable organ taken out in attempts to discover an organic cause before he, or in most cases she, is permitted to continue to the journey's end, without even a consultation with a neuro-psychiatrist.

The only thing that can be said in justification of this sad state of affairs is that the medical colleges have failed and continue to fail to give their students sufficient training to recognize the importance of psychological medicine. Even in the medical world we have the evil effects of improper training. Therefore, it ill affords us to be too severe with parents who are deficient in the knowledge of proper child training.

We physicians who specialize in neuropsychiatry are not immune to the same criticism, for we tell the other practitioners that the neuropath speaks a different language from his fellows, and he must not be taken seriously; yet, in our attempts to instruct the physician we use such technical and ambiguous terms that he fails to grasp our meaning. Frequently I suspect he concludes we are not altogether mentally normal ourselves.

As an illustration, we tell him that the process of instilling fear into a human mind begins at birth and results in an adult life filled with emotions so deeply suppressed in the subconscious that they are unknown to the individual and yet are often the cause of a mental breakdown. We then fail to explain satisfactorily what the subconscious is, why it is that we do not know or recognize these suppressed emotions in ourselves, and why they have no apparent relation to the symptoms presented, or perhaps appear in a disguised form.

There are some 400,000 legally adjudged insane persons in the United States today. The percentage of mental disease is higher than that of all other diseases combined, and there is no evidence to warrant the belief that the numbers are decreasing. A large per cent of these unfortunate people are insane as the direct result of improper training received during childhood. There is, perhaps, an even larger number of persons afflicted with so-called "nerves," due to the same cause, wrong beliefs, habits of thought or suppressions, or fears inculcated by parents, teachers and associates during childhood, which ultimately prevent their adjustments to the realities of life. Because illness is the most convincing and convenient excuse for failure to meet an unpleasant situation effectively, their unpleasant emotions are unconsciously concealed and often present themselves disguised as physical symptoms. There is hardly a disease that I could mention, outside of the list of febrile infections, which cannot be simulated by a disordered nervous system.

Again, these symptoms may be disguised as a resentment of the social code, conflicts with the laws of the land, or else in the establishment of habits which ultimately lead to a mental disintegration. In such a case the individual rationalizes or excuses his habits or acts by falling back upon alibis which are satisfactory to himself and sufficiently plausible to others.

In order to reduce the enormity of mental disease, no intelligent physician should be content merely to relieve the symptoms caused by nerves, but he should make every effort to disclose the underlying factor responsible for the appearance of the symptoms. Otherwise they will surely return, even though so thoroughly disguised as to mislead the attending physician into thinking they have no connection with the previous attack.

In May of 1907 it was my very great fortune to be associated with the late Sir Frederick Mott for a short period of time. He had not long returned from a trip to South Africa, where he headed a commission to study the nervous and mental disorders common to the savage races, and while journeying from London to Claybury, where his laboratories were located, he asked me what I considered to be the greatest etiological factor in the production of insanity. I had not given that particular subject much thought and told him so, but hazarded a guess that syphilis and alcohol were perhaps the two greatest factors. Much to my surprise, his answer was that the greatest of all single factors was civilization, and the manner in which we were reared. He did not, as I recall, use the phraseology common to the present day, but his meaning was the same: that it was the inhibitions, suppressions of our instinctive desires and the bottling up of our emotions demanded by civilization.

During our conversation he also told me that he had come in contact with a small tribe in South Africa who had never before met a civ-

ilized man and insofar as he could learn they could not furnish a history of any mental or nervous disorder occurring in their tribe or their ancestry, except those due to organic lesions resulting from an injury or arterio-sclerotic changes. Their habits were not unlike animals in that they never repressed any of their instincts or their emotions. In the semi-savage tribes he visited, the only mental and nervous ills presented were those due to the vices of civilization, syphilis, alcohol or drugs. The higher up in the scale of civilization he investigated the greater was the variety of nervous and mental disorders observed.

It was shortly after this that he began his study of the ovaries and testicles in dementia precox and he noted that in every well established case there is a marked primary regressive atrophy of the interstitial cells, together with a failure to function. Despite the fact that he failed to mention a connection with this and sex repression, in view of the well known fact that most cases of dementia precox occur in young people whose character is so exemplary as to cause them to be regarded as mothers' boys and girls, I strongly suspect he was not unmindful of the connection.

It would not be altogether unreasonable to suspect that other glandular changes might result from the fears, inhibitions and other emotional disturbances inculcated in children during their early years. One thing can be said with certainty, and that is that if we do not find physical outlets for our emotions and civilized equivalents for our instinctive desires, the ultimate future of civilized man is not very pleasing to contemplate.

The number of neuro-psychiatrists is entirely too limited to educate our lay population to the many dangers lurking in the present day methods of child training. However, we are of sufficient numbers to present not only the dangers, but the effects of mistakes in child training to our fellow physician, provided we speak his language.

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DISCUSSION (*Abstract*)

Dr. R. C. Bunting, Memphis, Tenn.—The avoidance and correction of neuroses and mild psychoses in children today primarily owes its origin to a group of men who some twenty-five years ago stressed the fact that the individual should be studied as an entity, through his environment and his reactions to his environment, rather than by physical study. In this group were such men as Southern, Meyer, Samson, and White. About 1909 Dr. William Healy began the application of these principles in the Psychopathic Institute in Chicago. It was from this group of men, and the subsequent studies of psychiatrists and psychologists, and I wish to mention particularly the child guidance clinic, which owes its origin to the Hartness Foundation, that we have our present day conceptions of the value of child guidance in the prevention of nervous and mild psychotic disorders.

The basic principle of mental hygiene is the early detection of mental and nervous disorders, and their correction, with the hope and belief that there may be a more normal mental life in later years. Is it not pertinent to recognize the necessity of training the parent, too, that he may know himself so well as to be qualified to guide and teach his child? It is in this particular that I wish to discuss Dr. Owensby's paper. There is no gainsaying the truth of the statement, that many parents do not know how to guide even a normal child, so any attempt in the direction of guiding one displaying personality problems, conduct disorders or what-not, has little hope of success; rather the particular case is made worse.

Perhaps the parent attempts to solve the child's problem purely from an intellectual approach. This is usually doomed to failure. A child's difficulty can be best understood if the parent understands that the child is merely reacting in its own way to his parents' overbearing attitude; to too much conflict between parents themselves; to a too ambitious study program laid down by the parent, or to an over-zealous parent who fears she sees in her child symptoms of a nervous disorder which she, herself, manifested at one time. This never occurs to the father and mother who are, in all honesty, solicitous of their offspring's mental health. Are there many parents capable of recalling the finer feelings of their own childhood, the results of their childish disappointments, nagging of parents, the strain of efforts to measure up to mother's expectations, or too great solicitation for the child's physical health and comfort? These over-solicitous parents undeniably are responsible for the development of neurotic symptoms in the child. No one can hope successfully to guide a child in mental hygiene if he does not know how to estimate the value of the child's emotions and reactions. Do not parents often attempt, unconsciously, to shape their child's conduct, both at home and abroad, to their own as it was in their youth, and when the child attempts to follow his own initiative, with a resulting different reaction to a situation, is the parent able to understand? Because of the dissimilarity he upbraids the child and there ensues, usually, a scene, if such nomenclature may be employed. It is true many children come through in spite of such misunderstandings, but it is also pertinent that many do not. These later develop mental and nervous disorders and, sooner or later, reach the physician, or others, for treatment.

In the home training of the child the parents' first duty is to understand children in general; second, to understand a particular child. The general knowledge of children tells the parent what the fundamental strivings are, from which some insight is gained as to the nature of motives for conduct. The knowledge of the particular child implies that the parent understands that the child has a mental life, has ambitions, joys, sorrows, fears and so on, and that these many emotions are aroused in the child just as they are in the adult. We are all familiar with the parent who, through a lack of general and particular knowledge of chil-

dren, has the perspective that never permits the child to grow up, so that when adult life arrives there is the absence of self-initiative, self-reliance and self-assurance. The child who inherits such parents has a handicap that is difficult to overcome.

Then it is incumbent upon the parents to see that the child is permitted, nay forced, to have contact with the outside, for, is not much learned of the problems of social adjustment from other children? Far more is absorbed from children than can be learned from adults. Letting the child face life as it is, meeting obstacles and overcoming them, facing his problems as he finds them, is what trains the individual, strengthens him and creates self-assurance and self-initiatives, and it devolves upon the parent to appreciate these necessities for child training.

A home containing children holds responsibilities as well as pleasures for the parents, and they should accept the responsibility for the development of habits and the establishment of adequate modes of conduct in the child that leads to an harmonious daily life. In the first and last analysis the duty of child training rests squarely upon the parents.

Dr. Walter Timme, New York, N. Y.—This problem of early training of the child connotes so many other collateral problems that the future looks to me to be highly complicated. In order to have the proper environment for the child and the proper teaching and education and guidance of the child, we must guide the parents, the judges, and the community. We have been trying to guide communities for ever so long with only moderate success.

When we say that we have to force the child to have contacts with the world so that that child may understand his position relative to the world, the very word "force" seems to me to be rather inapplicable to the child. As soon as you attempt to force an individual, he takes the other road. We see what trying to force this entire community into the prohibition camp has done. It takes the other road.

I sometimes feel that allowing a child to grow up as he will, without attempting to force him into ways that we think are best, is perhaps the wisest plan of all. I do not say that the plans that have been advanced here are wrong. My plan may be wrong. In fact, I have no plan, but I certainly am confused by what we are attempting to do. The problem is tremendous and when we have arrived at the end of it we have done what? We have substituted for a natural plan of evolution our plan; whether it is better or worse remains to be seen. We probably will not live to see its results.

If you will look upon the adventuresome spirits of this world, those that arrive somewhere, let us say in a monetary sense; if you study, for instance, the newcomers in the field of finance, you will see remarkable things that perhaps have not occurred to you at all in your work with child guidance.

These pragmatic successful examples that we have before us constantly are so different from those about whom we tell the children, that the whole question does not resolve itself at all; it leaves those that see the actual successful individual cases in a maze. I do not know where I am, actually, when I witness what we are trying to do, shipwrecked almost in the beginning by the actual doings of people who are brought up and who are bringing themselves up in an entirely different manner, with success.

It really, then, comes down to this: do we teach children ethics? I do not see that we do. Religion comes in; the minister, the rabbi, comes to our institution and says: "You teach these children everything else, but no religion. We have to infuse ethical religion into them before they will know what the difference is between right and wrong and have the courage to go to the right path."

Following upon that, we began to introduce some of the ministers and some of the rabbis to these children, with worse confusion than ever before, because each one had his own method of inculcating his religion, and each one could but show his meager success to the observant child whom he was trying to teach. With all our efforts to change a community, which we are trying to do, which perhaps is our aim, we are simply making, probably, huge strides toward state socialism. Perhaps we do not recognize it as such, but that is what we are doing. Now, is that an advance or is it not an advance? Is it going counter to the laws of evolution, or is it not? I do not know. That is a problem which is confronting me more and more year after year, and I really do not know where I stand.

It seems to me that the very effort that a child makes to overcome the difficulties of a bad home, a bad environment, is what will make him a good citizen later on, all the more efficient because of his early struggles against adversity.

Dr. Beverley R. Tucker, Richmond, Va.—One of the most hopeful things I have heard for a long time is the fact that Dr. Walter Timme has bent his mind on this subject and confesses he does not know and is still confused as to some aspects. That is the attitude all of us should take.

I regret extremely not to be able to agree with Dr. Owensby in his idea of the great importance of environment and in his idea of the negligibility of heredity. Personally, I think it is the old fight between the Lamarck school and the Mendel school, of which the elements of both are probably right.

We certainly do follow our parents in our characters and traits. We follow them in the shape of our features, in the texture of our skin, in the formation of our bones, in our nervous systems, and in our brain cell morphology. That following leads us to react and to do things in life in certain ways which no training can entirely counteract. All we do by correction, child guidance and child training is to do what the zoologist does. We can take the wild animal and put him in a cage or in a confine, and as long as he is in there he is harmless. As long as the teacher is the person who enters the cage he does not show his animalistic instincts and tear him to pieces, but he is a tiger all the same.

It would be almost Utopian if we could, by guidance, by studying this problem, by organization, by laying out certain chalk lines on which to walk, correct these subjects. The solution will come when the world ends, and only then. We can better things, we can keep these people confined, but if anybody can, by child guidance or any other means, prevent a paranoiac from developing paranoia, a dementia precox from developing dementia precox, or a manic depressive from developing manic depressive insanity, then I will certainly take my hat off to the man who can tell us how to do it.

Dr. Owensby (closing).—The scientific study of the training of the child and its effect on the conduct, health, character, success or failure to adjust to his environment during its adult life, is a comparatively new subject for research, and I am confident that it will

bring forth many valuable observations hitherto unnoticed. It is a human trait to overlook the things that are near us and to search for them in remote places, and we physicians are not immune to this fault. We are very prone to look to the endocrins, metabolic disorders, or other remote places for the causes of the peculiarities of human behavior, when in reality they are due to the improper training of the child which has in turn caused an emotional, nervous, or mental instability of such nature as to produce a dysfunction of the endocrins, metabolic disturbances, or other puzzling symptoms. The evolution of man from the savage state is the result of training and the demands resulting therefrom. The study of the results of the training of the child and the conditioned reflexes originating in childhood will be productive of greater knowledge than most men surmise.

REPAIR OF CICATRICIAL ECTROPION BY FREE DERMIC GRAFT*

By JOSEPH W. TAYLOR, M.D.,
Tampa, Fla.

In 1876 J. R. Wolfe, surgeon to the Glasgow Ophthalmic Institution, under the title, "A New Method of Performing Plastic Operation," reported two cases of lid repair by use of free dermic grafts from the forearm. In summing up his work he said:

"The pedicle has, in my opinion, been a source of great embarrassment to surgeons and the constant employment of it has tended rather to retard than advance the progress of plastic surgery."

Yet, as late as this year the writer on visiting one of the leading ophthalmic hospitals saw a small cicatricial ectropion (upper lid) repaired by going up on the forehead and bringing down a flap with a large pedicle. The surgeon, upon being asked why he did not use the free dermic graft, stated that he had never tried it.

In the literature there seems to be about an even division between those who favor a free dermic graft and those who advocate the epidermic graft. Either is easy to use and so satisfactory that I do not see the occasion for using the pedunculated flap in cases of cicatricial ectropion where the conjunctiva is intact.

Careful technic and good judgment, as in all operative procedures, are the secrets of success. Wolfe's wise conclusions were:

"If we wish a skin flap to adhere to a raw surface by first intention or by agglutination, we must be sure it is completely cleared of all areolar tissue and properly fixed in its new place."

*Read in Section on Eye, Ear, Nose and Throat, Southern Medical Association, Twenty-Second Annual Meeting, Asheville, North Carolina, November 12-15, 1928.

Wolfe's method was immediately taken up and operations for ectropion by the use of the detached flaps of true skin were reported by Wadsworth, Aub, Noyes, Howe, Benson and others. Partial success rewarded many of the early efforts, but contraction of the graft was annoying and there were many failures due to the fact that "agglutination" did not take place. Evidently improvement in technic was called for. In 1919 Dr. John M. Wheeler presented a paper entitled "Free Dermic Grafts for the Correction of Cicatricial Ectropion." In this and later articles he offered many suggestions and improved technic which successfully carried out the method propounded by Wolfe. He states that:

"Unfortunately, no method of treatment has been generally agreed upon as superior to others. In my mind the choice is made. For most cases of scar ectropion, transplantation of free dermic grafts offers by far the most satisfactory means of correcting the deformity."

He believes that skin characteristics are carried better by grafts made up of epidermic and true skin than can be possible by transplanting grafts of epidermis only, without the element of true skin; that Thiersch (epiderm) grafts are best suited to cases of severe burns of the face with consequent loss of the normal color, texture and characteristic appearance of the facial tissues.

Pedicle flaps often fail and not uncommonly give the patient a grotesque appearance. These flaps must be taken thick or life is imperiled. Thick flaps are manifestly undesirable for lid grafts. Passive congestion, which leads to the death of all or parts of many pedunculated flaps, is not to be considered in the use of dermic or epidermic detached grafts. Their life depends on contact with the underlying tissues and blueness and death from impairment of old circulation cannot result. Anemia in these grafts is soon followed by new vascularization and vitalization, provided proper contact with healthy underlying soft tissue is given.

In the use of pedicle flaps it is not possible with safety for the flaps accurately to match the needed shape, which is almost always crescentic or fusiform. The pedunculated flaps have a large and useful field in many plastic procedures, but little can be said in their favor for lid work. Wheeler makes the statement:

"If I mistake not, nothing has hampered the progress of plastic surgery so much as the idea that pedunculated attachment is necessary for the life and well being of grafts."

Wheeler, in a paper before the Academy in 1919, suggested three sites for the removal of

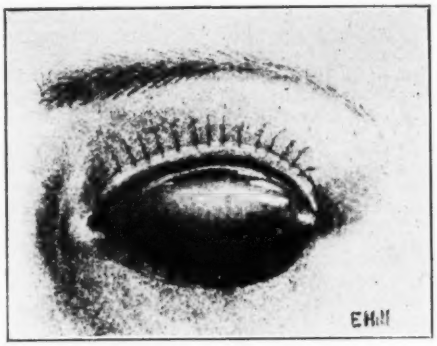

Fig. 1
Showing lid everted and grown to skin just beneath the brow. Note marked edema of conjunctiva.

true skin grafts. The inner surface of the arm, the temple and the upper lid. He finds the arm graft more difficult to prepare and slowest in assuming the characteristic of the surrounding tissues of the lids. It requires several months for the arm graft to take on the proper color, and in some cases it never does quite accurately match. The inlays from the temple match well in color and texture if the graft is not too thick. However, he finds the skin from the upper lid when available the ideal location for taking the grafts. They match in all characteristics from the start and after a few weeks it is almost impossible to see the outline of the graft.

Wheeler's Technic.—The aim should be total correction, even to proper conduction of the lacrimal secretion. The primary incision is parallel to the distorted lid margin. Cicatricial tissue must be painstakingly removed. Gentle handling of tissues is essential. Hemostats are used sparingly, and no vessels are tied. The lids are fastened together by making firm adhesions on either side of the pupillary area. The epithelium at corresponding points is dissected off, and mattress sutures bring the raw surfaces together, through small pads of rubber (cut from sterile rubber tubing). Later stretching allows of slight separation of the lids so that the patient can see between the margins.

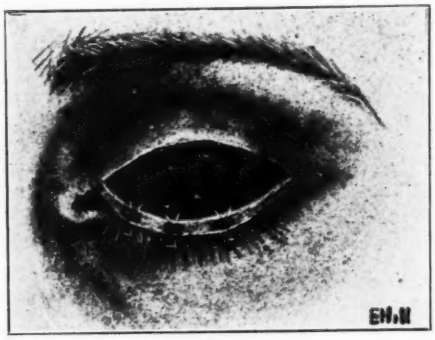

Fig. 2
Showing area prepared for inlay, all scar tissue having been removed and lids sutured together.

In removal of the skin from the upper lid, practically no allowance need be made for contraction. From the upper lid of a young adult 25x50 mm. of skin can be safely taken, and from an elderly person even more. No grasping forceps are used. The skin is outlined first and a cataract knife is slid under from below upward towards one end and then towards the other. The piece is picked up with the fingers and freed. The graft is then placed on a pad wet with warm saline and the fragments of subcutaneous tissue adhering are snipped off with scissors.

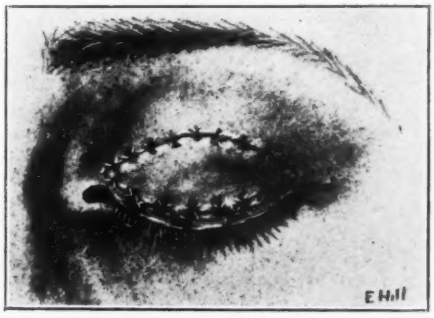

Fig. 3
Inlay in positions. Fine pariffin silk sutures used.

Fine silk sutures (No. 1 twisted, impregnated with paraffin) are used to stitch the graft in position. The ends are first sewed, then the margins. The wound in the upper lid is easily brought together and no undermining is necessary.

In cicatricial ectropion of an upper lid, skin from the fellow upper lid is used, never from a lower lid.

Dressing.—The graft is covered with two layers of gutta percha, the grain crossing each other, slightly smeared with sterile petrolatum. Gauze fluff is packed, secured by adhesive, and then a pressure bandage is applied. The good eye is independently bandaged and its dressing removed in three days. The operated eye is redressed after six days and the graft cleansed with damp (not wet) boric acid sponges, and

the sutures removed. The same type of dressing is continued, with changes every two days for another week, after which the graft is kept smeared daily with a little sterile petrolatum. Three weeks after operation massage is started with sterile petrolatum, and continued daily for several weeks. The adhesions between the lids are left for three months, then separated by scissors.

CASE REPORT

J. E. B., age 20 months, an American, the youngest of four children, was large for his age and in perfect physical condition.

Seven weeks previously he had fallen, producing an abrasion near the outer canthus of the left upper lid. This later became infected with abscess of the lid. It was opened by the family physician. He made an incision in the lid extending from the outer to the inner canthus. Erysipelas followed, involving the upper lid, forehead and temple. When I first saw the child there was complete destruction of the left upper lid except for one or two millimeters of the lid margin containing the cilia. The lid margin was adherent to the skin just below the eyebrow (as seen in slide). There was a great amount of edema of the exposed conjunctiva, but no involvement of the cornea. The edema well protected the cornea from exposure.

Under gas-ether the lid, with the cilia attached, was dissected loose. All scar tissue was carefully removed except on the area beyond the external canthus which did not affect the lid. There was such a large area of raw surface to be covered (20x30 mm.) that I hesitated to follow Dr. Wheeler's suggestion and take a graft from the opposite upper lid, especially since the cicatrix was keloid in character. It was decided to take a dermic graft from the opposite temple. The graft was taken slightly larger than the area to be covered. The size of graft was outlined and with a sharp knife an incision was made through the skin. It was then dissected free from the underlying tissue with scissors. All areolar tissue was removed and the graft was sutured in place, using fine paraffin silk. The lids were sutured together in three places. Vaseline and rubber tissue were used over the inlay, then gauze fluffs and a pressure bandage. Both eyes were bandaged separately. The next day the child fretted so, because both eyes were bandaged, that his temperature went to 103.5°. The bandage on the unoperated eye was removed and the temperature almost immediately returned to normal. The dressing on the operated eye was removed on the eighth day. The inlay had taken on a beautiful pink, healthy color. The sutures were removed and the lid margin did not adhere. As these inlays are quite stiff for the first few weeks or months, I did not think it advisable to suture the lids together again in a second attempt to get them to adhere. Vaseline dressing was used and changed every second day for one week longer. At this time gentle massage was used. The lid remained stiff and hard for at least two months. I do not believe anything was lost by the lid's not being sutured during this period. This brings up the question whether it is necessary to suture them where a free dermic graft is used (upper lid) and if so, is it necessary for so long a period as is customary? At the end of four months a fold could be seen beginning to form in the lid when opened. At present, after eighteen months, the child has a very presentable and useful eyelid.

Fig. 4
Results a few months after operation.

In conclusion I should like to stress the ease with which these cases can be handled and the satisfactory results obtained by following Wheeler's technic.

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DISCUSSION (*Abstract*)

Dr. W. D. Gill, San Antonio, Texas.—In reconstructive surgery of the lid there is no doubt that the free dermic graft according to the method of Wolf has provided a very excellent operative measure.

Certain pre-operative considerations must be observed. If the defect of the lid has not entirely healed and if we are likely to have a residual infection, we must consider that, and if there is residual infection in the lid we must obtain sterilization. This can be accomplished by chlorazene paste or dichloramin in oil. Dakin's solution cannot be used.

The first essential is mobilization of the lid by excision of scar tissue. If we over-correct, so much better. Shrinkage will take care of that. Hemostasis is absolutely necessary but we cannot employ ligatures. An accurate fit is essential, and if we do not wish to take skin from the temple or the arm, or the other eyelid, that from the shaft of the penis is about the

right thickness and serves very well for a free graft.

We must provide an exit for wound secretions by the use of interrupted sutures, by providing a straight stab wound, or by nicking the center of the graft.

The utmost care is necessary in handling the graft. We must protect it from thermal, chemical and mechanical insults. Pressure is necessary; immobilization of the lids is necessary. We certainly cannot have success without these. I think the three months' period is perhaps the shortest that we can use intermarginal fixation.

Intermarginal fixation is used for several reasons: first, to provide a flat surface for the graft to rest upon; and, second, to keep the lid in a quiet condition over a period of time. Scar tissue does not obtain the maximum degree of contraction for at least three months.

As a general rule, change of dressings in forty-eight hours is soon enough. Then we can tell whether the graft is going to take by its color. If it is pink it is vital; if it is purplish there is no hope of its taking.

The post-operative treatment is very important, and among these cases there are certain traumatic ones where debris may be locked up in the scar tissue. We must not overlook giving such patients tetanus antitoxin. This is particularly important in cases that come from automobile or street car accidents.

Physiotherapy treatment later is valuable. Where the graft does not regain its normal color as rapidly as we think it should, we can use radiant heat. Massage is indicated if contraction of scar tissue is vitiating the result of our operation. We may overcome redundancy by the use of pressure appliances.

Dr. Calhoun McDougall, Atlanta, Ga.—I was associated with Dr. John M. wheeler at U. S. Army Hospital No. 2, Fort McHenry, Md., where much of the pioneer work in skin graft surgery of the orbit and surrounding tissues was done. Preparation of the field is very important. We have had very few infections as a result of our technic and we often removed the lacrimal sac before doing the graft. We can remove a large area from the other eyelid without causing a deformity, and usually removed one-third larger sized graft than the area to be covered. We very carefully measured the area and then made the graft one-third larger. Also in preparation for the graft Dr. Wheeler insisted on putting the graft on the palm of the hand and gently cutting out the dermic tissue with Stevens scissors until the skin graft is very thin. This is called a true dermic graft. If you use a graft from the arm or the temple this is more necessary than a graft from the other eyelid. If this graft is properly prepared and thinned down it will take more readily than a thicker graft.

Dr. Wheeler's idea in taking the graft from the temple was to secure a graft that would match in color the surrounding tissues. We had many cases with powder burns all over the face and eyelids. The graft taken had black powder spots also and matched the surrounding tissue. We also had some of the grafts exposed to the sun daily in order to get a better cosmetic effect.

We often used dermol instead of waxed silk for sutures and it worked very well. We did not use drainage, but used interrupted sutures, and very few were infected.

At the first two or three dressings we often noticed an apparent sloughing of the graft and became disappointed in thinking that it was not taking, but later the vascularization of the graft could be seen and a great majority were successful.

GOLD SODIUM THIOSULPHATE IN MACULO-ANESTHETIC LEPROSY*

By J. L. KIRBY-SMITH, M.D.,
Jacksonville, Fla.

Gold sodium thiosulphate has been used extensively during the past year in the treatment of lupus erythematosus and no doubt all of us are familiar with the results of using it in the treatment of this disease.

Last February a rather unusual case of ma-

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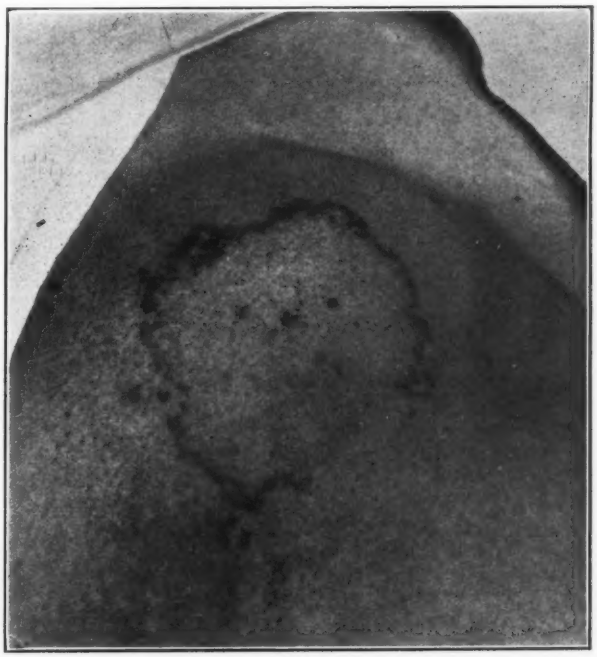

Fig. 1
Maculo-anesthetic leprosy lesion on left buttock.

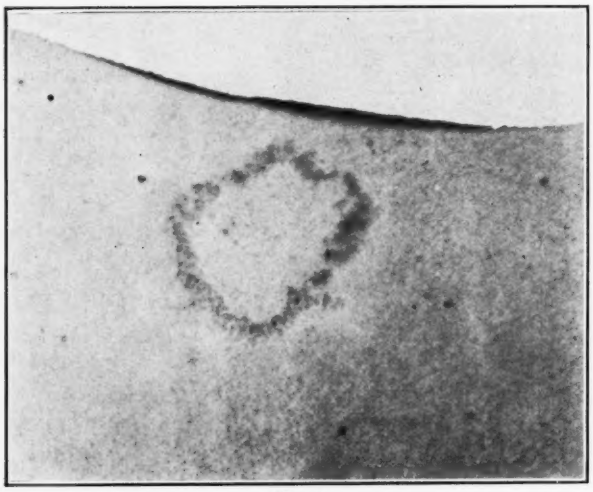

Fig. 2
Maculo-anesthetic leprosy lesion on the middle of the back.

culo-anesthetic leprosy came into my hands. Having in view the usual unsatisfactory results of treatment of leprosy, I decided to give this patient intravenous treatments with the gold salt.

In this short report I am making you today, attention is called to the very prompt and noticeable improvements both objectively and subjectively. Unfortunately the final reports of the treatment were not obtained. This report is made merely to show the effects of six intravenous injections of gold sodium thiosulphate.

I thoroughly understand that from time to time various remedies have been suggested and tried out in the treatment of leprosy. Oftentimes various drugs have produced apparently curable results, but with time and further efforts these remedies have been proven to have no definite curative effects.

A clinical diagnosis of maculo-anesthetic leprosy from a dermatological point of view in the case under discussion could not be disputed. Nasal smears were reported negative on two occasions by the State Board of Health and biopsy was refused by the patient, but those of you who are present today, on seeing the pictures I shall show you, will not question for a moment that the diagnosis was correct.

Mrs. E. J. B. consulted me on February 4 for the treatment of a skin condition that had been in existence for ten years. This lady was 25 years of age, she had been married five years, had a child of four, apparently normal, and her husband was apparently normal and in good health. Physically this woman was a normal, healthy female. Her weight was about normal, general health good, menses regular, and there was no complaint other than that referred to.

The blood Wassermann was negative, urine negative, heart, lungs and other vital organs negative. A thorough examination of her skin revealed three active lesions that were absolutely and indisputably leprosy. On her left buttock and in the middle of her upper back were two circinate anesthetic lesions, the central part of which was white and lustrous, the outer borders of which were slightly raised and of a brownish pigmented character. The palmar aspect of the left hand showed a faint pigmented condition. The tips of the thumbs, the second and fourth fingers showed slight trophic ulcerations, the nerves of both ulnar arms were very much involved, the left particularly so. Palpation of one of these nerves showed it to be as large as an ordinary lead pencil.

I decided to give the gold sodium thiosulphate (Abbott's gold thiosulphate

Fig. 3
Maculo-anesthetic leprosy, showing pigmentation in the palm of the hand and trophic ulcers of the fingers.

was used). On February 7, 100 mg. of gold were given intravenously. This dose was repeated on February 9 and 11, and on February 21 the patient made a report showing marked improvement both objectively and subjectively. The trophic ulcers on the tips of the fingers were completely healed, the numbness in the hand had improved 100 per cent, and the lesions on her body improved in that the outer borders were less noticeable. On February 21 another 100 mg. injection was given, and again on February 23. After these six treatments the patient returned home and reported on March 3. She was given another injection of 100 mg. of the gold sodium thiosulphate. At this visit, which was made one month from the first visit, a marked improvement had been obtained both objectively and subjectively in the active lesions of leprosy that were present on the first visit. From the time that I first saw the patient, the matter of reporting her to the health authorities had been before me; but owing to two negative reports of smears from the nares and because she refused to have a biopsy performed, I thought of calling in the proper authorities to verify my clinical diagnosis. However, after I discussed the matter with the patient, gave her the diagnosis, and told her what could be expected in the way of a permanent cure, she failed to return, and although I made a number of inquiries, she could not be found.

Greenleaf-Crosby Building.

DISCUSSION (*Abstract*)

Major O. E. Denney, Carville, La.—We have had very little experience with gold sodium thiosulphate in treating leprosy. We have experimented with sodium thoisulphate in some of the acute manifestations, with but little encouragement. There have been a number of reports from various parts of the world in the past few years and there seems to have been quite a difference of opinion as to the value of this drug. We have nothing to report at the present time.

Dr. J. N. Roussel, New Orleans, La.—I have tried everything in the pharmacopeia for leprosy and have seen the patients improve with practically all the remedies. My experience has been that almost any intravenous medication will bring improvement, but I assure you that it does not last long. I have over 25 cases of maculo-anesthetic leprosy that have cleared up completely under anthrax vaccine. I am sure nobody could have found anything the matter with them. That happened between 1920 and 1922. At this time the condition has recurred in all of them and I think most of them are worse now.

A long time ago Dr. Crocker, of London, reported that hypodermic injections of bichlorid of mercury would clear up the tubercular lesions, but that they would almost immediately recur and where the patient had one lesion before, he had one hundred after the treatment. The same thing is true of the anthrax vaccine. The lesions cleared up completely in about six or eight months, so that not a lesion was in evidence, but in less than ninety days from that time where there had been one lesion there were more than a hundred. It seems to me that the more we do for the cases the worse off they are. I have become quite discouraged and usually tell these patients that there is a gentleman at Carville who knows more about this condition than anybody else, and advise them to see him.

Dr. Kirby-Smith (*closing*).—Major Denney has unlimited opportunities to see results of the various remedies that are suggested for the treatment of leprosy at his hospital at Carville, and I realize that a remedy may induce temporary improvement in this disease, but, on further trial, be found to be insufficient. I feel there is no doubt about the benefits of gold sodium thiosulphate in the treatment of cutaneous leprosy. In the case that I have reported, I gave an unusually large amount of the gold salt intravenously. I gave this patient 250 mgs. in seven days. I had her under my care for over six weeks. When she came to see me she could not use her hand. The painful trophic ulcers of the end of her fingers and the discoloration in the palms of her hands were completely eliminated when she left me. This case was reported for the interesting result of a new line of treatment.

CONGENITAL HYPERTROPHIC PYLORISTENOSIS*

By JACKSON W. LANDHAM, M.D.,
Atlanta, Ga.

There is no more interesting or spectacular condition of early infancy than congenital pyloristenosis. The infrequency of the cases and lack of operative and autopsy findings induced conservatism in the management of these patients for some time after the report of the first case of pyloric obstruction in infancy by George Armstrong, a London physician, in 1777. The description of this case indicates that it was pylorospasm. Hezekiah Beardsley reported the first case of pyloristenosis that was confirmed by autopsy in 1788. Hirschsprung reported five cases before the German Pediatric Society in 1887. Soon after this cases were reported in the literature of all countries and more radical measures of treatment were employed.

The first operation for pyloristenosis was a jejunostomy done in 1893, which was followed by death of the patient. Prior to 1904 there were only five cases operated upon in the United States, with a mortality of 100 per cent. These operations were gastro-enterostomies and necessarily required much time in their performance. From 1904 to 1914 gastro-enterostomies in a large number of cases were reported by Scudder of Boston, Downes of New York, and Richtor of Chicago, with a mortality of about 50 per cent.

The cause of congenital hypertrophic pyloristenosis has been the subject of many theories. The information that has been obtained by operative and autopsy findings has limited the

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Fig. 1, Case 1
Pylorospasm. There is slight cardiospasm present and the peristaltic waves are very irregular. A considerable quantity of the opaque meal has passed through the pyloric antrum.

accepted etiological factors to two. One theory is that there is a primary hypertrophy of the pyloric sphincter. The other is that the condition is primarily spasmodic due to some gastric irritation and that the hypertrophy of the pylorus is secondary. That the condition is congenital in many, if not in all cases, is indicated by the presence of marked hypertrophy of the pylorus found at autopsy on infants born prematurely and those operated upon during the first few days of life. There is, however, a difference in the degree of hypertrophy and obstruction, because some of these infants manifest no symptoms until they are four to six weeks old. The condition occurs more often in male babies, the ratio being about four males to one female, and the first born is more often the victim than are the other babies. In the literature consulted, race is not mentioned as an etiological factor. It is interesting, however, to know that there is not a case of congenital pyloristenosis on record during the last five years in the colored section of the Grady Hospital. It may be that it does not occur in the negro race.

The first and most constant symptom of congenital pyloristenosis is vomiting. This may begin during the first few days of the child's life, or it may not appear until the child is four to six weeks old. In 50 per cent of the cases the vomiting starts during the first two weeks of life. At the beginning it may not differ in type from the vomiting caused by other disturbances, but it soon becomes explosive or projectile in character. The child may vomit after each feeding, or it may retain several feedings and then vomit the entire contents of its stomach. Very soon after the onset of vomiting visible gastric peristaltic waves can be seen and frequently a tumor about the size and shape of an olive can be palpated in the region of the pylorus. There is rapid loss in weight, accompanied by dehydration. Constipation is an important symptom. The appetite is good and the child is always ready to take another feeding immediately after it has vomited.

The post-mortem and operative findings show no evidence of an inflammatory process. There is a marked fibrous thickening of the circular fibers of the pylorus, frequently amounting to two or three times the normal thickness at this point. There is no thickening of the mucosa, but it is often puckered and thrown into folds which increase the degree of obstruction. In neglected cases the stomach is usually dilated and there may be some dilatation of the esophagus.

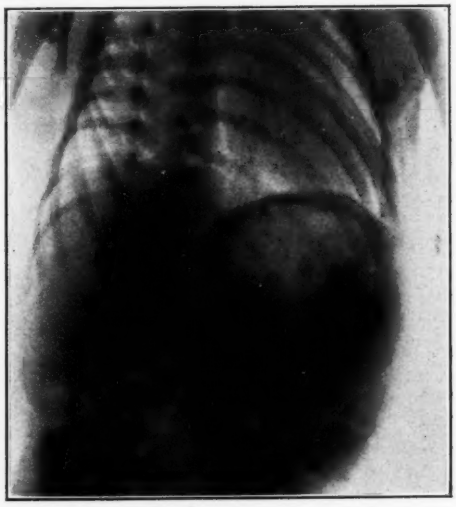

Fig. 2, Case 2
Pyloristenosis in white male, aged 5 weeks. There is vigorous peristalsis but none of the opaque meal was forced through the pyloric antrum during the entire period of study.

Fig. 3, Case 3

Undiagnosed case of congenital pylcristenosis in a white male, aged two months. Baby was markedly dehydrated and was running an inanition temperature of 104° F. Ncte gastric atony and impaired peristaltic tone.

An accurate history is an important aid in the diagnosis. This will usually show that an apparently healthy male child, frequently the first born, has been seized with projectile vomiting during the first few days or weeks of its life. There soon develops a visible gastric peristaltic wave which is more evident soon after feeding. There is constipation and rapid loss in weight. A tumor can often be felt in the pyloric region during, or soon after, feeding. Congenital pyloristenosis should be differentiated from indigestion, cyclic vomiting and pylorospasm. In indigestion the child shows signs of more discomfort and there are evidences of indigestion in the material vomited and in the stools. Cyclic vomiting usually occurs in infants of neurotic parents and is not explosive in type. There are frequently explosive or projectile vomiting,

Fig. 4, Case 3

Same patient as Fig. 3 three weeks after operation. The opaque meal passes through the pyloric antrum without evidence of obstruction. The peristaltic tone is good.

Fig. 5, Case 4

Congenital pyloristenosis in white female, aged six weeks, when x-ray study was done. There were visible gastric peristalsis, projectile vomiting and marked constipation present. None of the opaque material passed through the pyloric antrum during the study.

visible gastric peristalsis, a palpable tumor, constipation and rapid loss in weight in pylorospasm which makes it impossible to differentiate this condition from congenital pyloristenosis without resorting to an x-ray examination of the stomach. This examination, which should always be made by means of the fluoroscope, requires a limited amount of time due to the fact that the babies are always hungry and that active peristalsis will begin immediately after the patient has taken the opaque meal.

Straus, Abt and Ahrens evidently depend upon

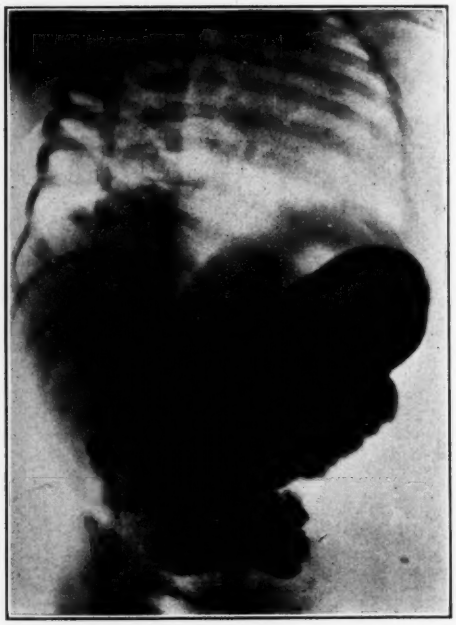

Fig. 6, Case 4
Same as Fig. 5 eleven weeks after a modified Rammstedt's operation had been performed. The peristaltic tone is good and there is no evidence of obstruction at the pylorus.

a film method of x-ray study, stating that if there is 50 per cent gastric retention at the end of four hours the case should be treated surgically, but if there is not over 30 per cent retention the patient should be treated medically. This method of examination cannot be relied upon, because in most cases of pylorospasm and pyloristenosis partial or complete vomiting of the contents of the stomach occurs before the four-hour period has elapsed. If the vomiting of the opaque meal has been complete before the end of the four-hour period the study will be of no value, and if the opaque material has been partially vomited there is no way of estimating the percentage of gastric retention. Since the information obtained from a film study of the stomach is limited, a fluoroscopic examination started as soon as the baby has been given the opaque meal and continued at intervals over a period of 15 to 30 minutes is the only accurate method of arriving at a differential diagnosis. Notwithstanding the accentuated peristaltic tone that is always present in pyloristenosis, none of the opaque meal is forced through the pyloric antrum during this period of study. In pylorospasm, which is usually accompanied by cardiospasm, the spasm is broken at intervals during the study and some of the opaque material passes out of the stomach.

The prognosis depends upon an early diagnosis and surgical treatment skillfully carried out before the baby has lost much of its weight. The mortality rate is increased sixfold when the child has lost 20 per cent of its weight.

The treatment of pyloristenosis is surgical, the Rammstedt operation or some of its modifications being the method of choice. If the diagnosis has not been made until the child has become markedly dehydrated and has evidence of acidosis it is better to administer saline and glucose by rectum and intraperitoneally for twenty-four hours before the operation. Rozar has reported two cases operated upon in which the wound of incision did not heal primarily, evidently due to the presence of marked dehydration of the tissues. After the operation the baby should be fed at three-hour intervals, according to the plan outlined at the Babies' Hospital, New York City.

SUMMARY AND CONCLUSIONS

(1) Great credit is due Armstrong, Beardsley, Hirschsprung and Thompson for their studies which resulted in a diagnosis of congenital hypertrophic stenosis of the pylorus as a clinical entity.

(2) Sufficient information has been obtained by autopsy and operative findings to prove that there is some unknown etiological factor that produces a stenosis of the pylorus at or before birth.

(3) The differential diagnosis can easily be made by fluoroscopic x-ray study of the stomach.

(4) The treatment is surgical in all cases of congenital pyloristenosis, the Rammstedt or some of its modifications being the operation of choice.

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DISCUSSION (*Abstract*)

Dr. Robert Drane, Savannah, Ga.—I feel from reading of the work of Dr. Dowan of New York, who operated upon 300 cases with a mortality of less than 8 per cent, after four weeks' delay, that surgical procedure is the proper method of treatment. Theoretically, from the fact that post-mortem shows there is a hypertrophy in a premature child, hypertrophy of the circular fibers is primarily at fault. The trauma of hyperperistalsis causes some edema of the mucosa, and that edema acts as a valve and prevents emptying.

In a case with the characteristic vomiting, stools, and tumor, I feel an x-ray is not necessary, though it is well to have it. Where there is not a tumor formation, but a generalized hypertrophy, making more of a band than a tumor, an x-ray examination is necessary.

Dr. Pierson has just told me of a series of cases that he has done on normal children. We feed infants every two to three hours and expect their stomachs to empty between feedings. Given barium, a normal child has not emptied his stomach in two to three hours. It takes about five hours completely to empty it. After an x-ray to test the amount of emptying, we must consider whether they vomited, how soon they vomited, and rely on the amount of barium which has passed the duodenum.

The chief factor which confuses us is pylorospasm. In this condition very little barium passes within an hour. Then the spasm relaxes and there is normal emptying. Therefore, if during our examination we see at times a normal emptying for a few minutes under the fluoroscope or in serial films, we can exclude stenosis. I feel, though, that we do our patient the most good if we operate early, before he is dehydrated, and before he loses too much weight.

I am sorry that in Savannah we neglect the negroes. The chronic cases come through the charity clinics and are worked up fairly well. The acute cases hardly ever come to us. I have not personally seen this condition in a negro child.

Dr. J. W. Pierson, Baltimore, Md.—I wish to elaborate upon one point made by Dr. Landham regarding the marked spasm of the stomach in the presence of congenital stenosis of the pylorus. The hypertonicity of the stomach is governed by the stage of the lesion in which one happens to observe it. In the early stages we see a marked spasm as described by Dr. Landham; but in the later stages, after the pylorus has become very thick and the muscles of the stomach have lost their tone, we see a large, dilated stomach. You will remember in the third and fourth films shown by Dr. Landham there was a large, dilated stomach in which the diameter of the pyloric end of the stomach equalled that of the cardiac end. This is, as we all know, the exact opposite of the picture produced by a spasm.

The effects produced upon the stomach by this condition are analogous to those produced by a duodenal ulcer. We are familiar with the marked spasm and hypermotility of the stomach in the early stages of a duodenal ulcer and we are likewise familiar with the large, dilated stomach in the later stages of a duodenal ulcer associated with adhesions. We must continually bear in mind this apparent paradoxical behavior of the stomach when we are diagnosing any lesion which involves the pylorus.

Dr. Landham (closing).—I would like to ask if any of the radiologists present has seen a case of pyloristenosis in a negro baby. The presence of it in the negro race is not mentioned in any of the literature that I have consulted and it is highly probable that it does not occur in the negro.

Of course I do not agree with Dr. Drane in his statement that in most cases of pyloristenosis the diagnosis can be made without an x-ray examination. Many unfortunate delays have been made in trying to make a differential diagnosis without x-ray examination. The clinical triad of symptoms, visible gastric peristalsis, projectile vomiting and marked constipation, are sufficient to demand an immediate x-ray study. These symptoms frequently occur in pylorospasm, but it is not difficult to differentiate stenosis from spasm. A delay made in trying out various feeding formulae, antispasmodics and waiting to elicit the presence of a palpable tumor in the region of the pylorus is certainly not justified and always is followed by marked loss in body weight, dehydration, and gastric dilatation and atony. My plea is for an early diagnosis in these cases so that the surgical mortality may be reduced.

APPLIED ANATOMY OF THE BACK IN RELATION TO INJURY*†

By R. W. BILLINGTON, M.D.,
and
R. S. CUNNINGHAM, M.D.,
Nashville, Tenn.

The anatomical approach to the question of back injuries is clearly established only in the more severe conditions. In mild or chronic traumata, the anatomical investigations are naturally limited by the routine survival of the patients. Nevertheless, the exact relationships are very important, inasmuch as, upon their proper understanding, depends the therapy rational to each case.

Since the understanding of the pathological anatomy of injury cannot be established by direct observation in the majority of cases, but depends rather upon rationalistic interpretation of signs and symptoms, it behooves us to visualize very carefully those normal anatomical relationships which become modified by disease and trauma. In our consideration of the back, we will refer to the vertebrae, the inter-vertebral discs, the articulations, the sacro-iliac synchondroses, the spinal cord and branches (cauda equina and all of the spinal nerves), the blood vessels and the supporting ligaments and muscles. We assume that the region referred to as the back includes the dorsal, the lumbar, and the

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†From the Departments of Orthopedic Surgery and Anatomy, Vanderbilt University Medical School.

sacro-iliac regions, remembering in this connection that injuries in these regions often cause physical signs and symptoms in other portions of the trunk and in parts or all of the lower extremities.

In considering the spine as a whole, it must be looked upon as a flexible pillar capable of considerable degrees of movement in every direction. This movement is facilitated by the nature of the inter-vertebral discs; the incompressible semi-fluid center of each of these structures, acting as a ball on which the rest of the disc plays. This would, therefore, constitute a universal joint were there no restraining mechanism. This mechanism, as is well known, consists of the articulations of the vertebral processes and the extensive ligamentous and muscular superstructure.

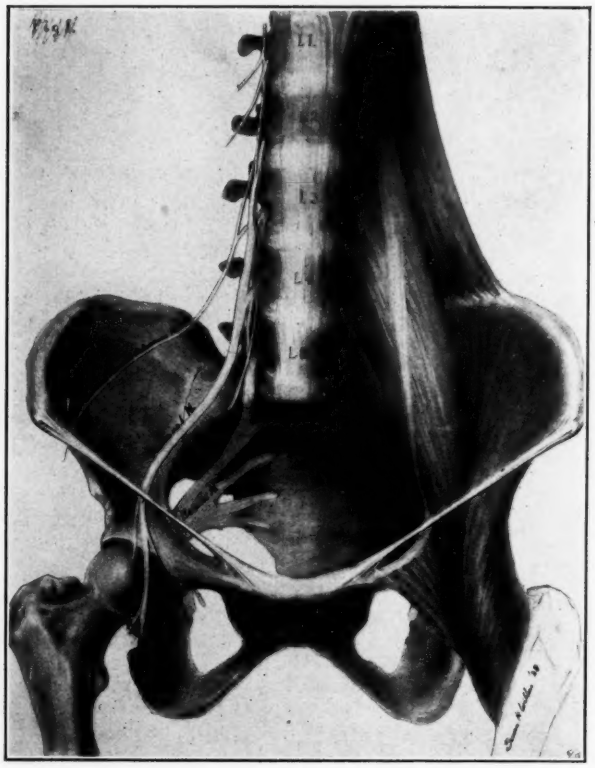

Fig. 1

Anterior view of pelvis and lumbar spine, showing the lumbar and sacral nerves as they emerge from the intervertibral openings. Note that none of the nerves is very closely related to the sacro-iliac joint. (The femoral nerve appears adjacent to the joint, but is separated from it by the iliac muscle.)

The motion involved in the spine as a whole is essentially the resultant of the many small movements occurring at the inter-vertebral joints. This flexibility is modified by certain peculiarities: (1) the motion is not equally distributed owing to the varying thickness of the vertebral bodies and of the discs; (2) the shape of bodies of individual vertebrae is such that motion is easier in certain directions than in others; (3) the spine as a whole has certain normal curvatures which modify the movements super-imposed upon these curvatures; (4) the motion of the spine is always modified by the possible limits dictated by the articulation of the articular processes and by the elasticity of the ligamentous and muscular supports. In considering these limitations it is obvious that flexion and extension are less limited in the cervical and lumbar regions. Specifically, flexion is more limited than extension in the neck, because the laminae interlock more readily. In the lumbar region, on the other hand, flexion is more extensive owing to the wide separation of the last two thoracic vertebrae.

It is well known that a flexible pillar, in which motion is possible in all directions, will, if subjected to two forces, operating in different directions at the same time, undergo torsion. This effect is obviously operative in many conditions in which the natural antero-posterior curvatures of the spine are modified by some factor producing a lateral bending.

For the purposes of this paper it has seemed to us wise to discuss the various traumatic lesions, both those definitely proven and those assumed from symptomatology, together with the anatomical features which seem to have an important bearing on the production of such lesions and which explain as many as possible of the various signs and symptoms observed. The conditions we have selected for discussion are (1) fractures, (2) dislocations and subluxations, (3) strains, sprains and ligament ruptures, (4) muscle injuries, (5) hematomas, (6) traumatic arthritis and synovitis, (7) compression and contusions of the spinal cord and nerves.

Fractures.—These may be caused by direct or indirect violence, more commonly the latter. Gunshot and other penetrating wounds may be associated with fractures of any of the portions of the vertebrae. The transverse processes of the lumbar vertebrae may be detached by muscular action or by a direct blow. The most frequent type of fracture is said to be that of the vertebral body due to compression when the trunk is forcibly flexed. This is most common in the dorso-lumbar region because, (1) this region is near the middle of the column to which the bending force is applied, (2) it has not the supporting effect of the chest wall above, and (3) the vertebral bodies in this region are smaller and probably have less resistance to crushing than those of the middle and lower lumbar regions. The fractures referred to above are usually easily recognized if clear radiograms (both antero-posterior and lateral view) are obtained and carefully studied. Injuries above the level of the first lumbar vertebrae are apt to be associated with serious cord injury and total paralysis, where as fractures at or below this level usually result in only partial paralysis, or none at all. Fractures of articular processes are seldom recognized, being difficult to visualize in the x-rays, but a consideration of the anatomy of these, especially in the lumbar region, would indicate vulnerability of these structures from extreme torsion and lateral bending of the trunk. Since these structures form the posterior boundaries of the inter-vertebral foramina, it is easy to see how their fracture may produce nerve-root pressure by displaced fragments or resulting hemorrhage, and swelling in the surrounding tissues. We believe it probable that such fractures, especially at the lumbo-sacral articulation, are more frequent than is recognized. Fractures of the sacrum and adjacent ilia are usually the result of severe direct crushing force rather than of indirect trauma, except fractures of the superior articular facets (processes) of the first sacral vertebrae. It is easy to see how one or both of these processes may be broken off by a forward shearing force exerted by the inferior articulating processes of the fifth lumbar (which may likewise be fractured) as in falls and from lifting heavy weights in the stooped attitude; also from torsional strains when the body is forcibly twisted on the pelvis. Other fractures of the sacrum and those of the wings of the ilia are usually easily diagnosed, heal readily, and are not important to this subject except as the sacro-iliac joints may be damaged. This is referred to elsewhere in this paper.

Dislocations and Subluxations.—Complete dislocations of the thoracic and lumbar vertebrae

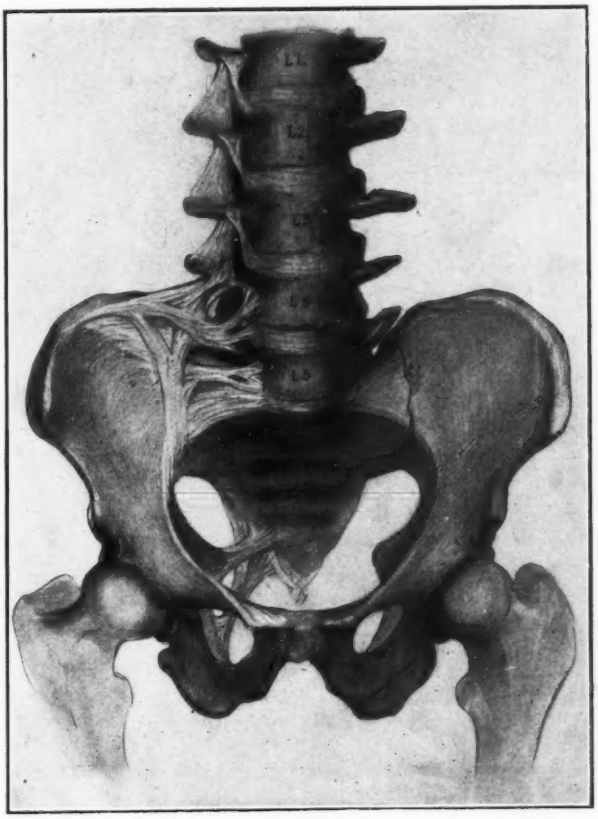

Fig. 2

Anterior view of pelvis and lumbar spine, showing on the right side the ligaments about the intervertebral openings. Note especially the small size of the opening through which the fifth lumbar emerges. On the left side, the close approximation of the fifth lumbar process to the sacrum and ilium is shown.

Fig. 3

Lateral view of pelvis and lumbar spine showing the openings through which the nerves emerge. These openings are bounded anteriorly by the vertebral bodies and the intervertebral discs, posteriorly by the ligaments and the articular facets, above and below by the bony processes. The extensive ligamentous connection of the various processes are also shown. These relationships indicate how sprains and tears of the ligaments may involve the nerve roots.

are rare and result from extreme violence. Partial dislocations or subluxations in these regions are more frequent and are nearly always associated with vertebral fractures, a fact easily explained by the anatomical arrangement of the inter-vertebral joints and their supporting ligaments. There is, however, one notable exception to this in the condition known as spondylolisthesis, or forward displacement of the fifth lumbar vertebrae on the sacrum. This is not of rare occurrence, but is often overlooked due to failure to get good lateral view radiograms of this region and to unfamiliarity with the characteristic posture and deformity of the spine which it produces. Here again a knowledge of the anatomy readily explains the occurrence and location of this displacement. It will be noticed in the articulated skeleton that the articular processes with their facets facing antero-posteriorly, are the chief structures, together with ligaments, resisting such a displacement, and that the oblique direction of the superior surface of the sacrum and the resulting lumbo-sacral angle necessarily throw great strain on these structures. Moreover, it is well known that anomalies of these two vertebrae (fifth lumbar and first sacral) are extremely frequent and that the shape and size of their articulating processes vary greatly. If these processes are unusually small they may be rather easily dislocated, with the spine flexed, without producing a fracture. As before stated, these articular processes at the lumbo-sacral joint are also probably often fractured, thus rendering subluxation comparatively easy.

Marked separation and displacement at the sacro-iliac joints occur rarely and are due to extreme violence and usually associated with fracture of the pelvic girdle. We have seen, and Hodgson has reported, fractures of the ilium and of the sacrum near the sacro-iliac joint, with no dislocation at this joint and with no symptoms even of strain of the joint resulting after healing of the fracture. This would seem to be evidence of the security and strength of these joints. This strength of the sacro-iliac articulation is very obvious when one studies its anatomical arrangement and tests the supporting ligaments in the cadaver. Yet we still hear much of sacro-iliac strains and subluxations as a cause of backache, and it is still a common belief that all one has to do partially to dislocate one of these joints is to stoop over or get out of bed carelessly. We agree with O'Reilly, and George and Leonard that subluxations of the sacro-iliacs can rarely if ever be positively demonstrated by x-rays. In fact, we have found no positive or convincing proof that they occur except from extreme violence, and then rarely. With modern x-ray facilities, there is no reason why displacements at the inter-vertebral and sacro-iliac joints cannot be demonstrated when they in reality exist.

Strains, Sprains and Ligament Ruptures.—The term strain is generally applied to mild back injuries and, due to the teachings of Goldthwait and others, "sacro-iliac strain" is a

term which has come to be applied to all sorts of back pains, genuine and pretended, largely supplanting the older and less impressive terms, "railway spine" and "lumbago." One of the writers (R.W.B.) discussed this subject in a paper on "Low Back Pains," at the Chattanooga Meeting of the Southern Medical Association in 1922, and we shall not attempt a full discussion here, though we could repeat practically every statement made at that time, with even stronger belief in their correctness. Suffice it to say again that "while it cannot be denied that traumatic lesions of the sacro-iliac joint do occur, a careful consideration of the anatomy and mechanics of motion and body strains shows a much greater liability to bone, joint and ligament injury in the lumbar spine than in the sacro-iliac joints." The lumbar and lumbo-sacral joints are much more movable and less securely supported and, with the powerful leverage that takes place in extreme movements or sudden twists, unguarded at times by the trunk muscles, they are very liable to ligamentous strain or rupture or even fractures of the small articular processes.

While it has not been demonstrated just which ligaments are most easily and frequently damaged, our studies of the anatomy and the application of forces leads us to believe these to be the ilio-lumbar ligaments and the inter-vertebral ligaments attached about the articular processes, especially those between the fourth and fifth lumbar and the fifth lumbar and first sacral vertebrae. This corresponds to the local tenderness and pain and to the nerve roots involved, in cases having referred pains, in the great majority of cases. The proximity of these ligaments to the inter-vertebral foramina and the resulting hemorrhage and swelling about the nerve roots in case of severe sprains rationally explain the sciatic and other referred pains often present. The fact that these referred pains often develop gradually, several hours or days after the injury is further evidence of nerve pressure from hemorrhage and swelling.

Muscle Injuries.—Contusions from direct violence and rupture of fibers from active muscular contraction may reasonably exist in many cases. We should expect the resulting swelling in the muscle if extensive to cause nerve pressure.

Clinically these cases might easily simulate ligament injury though we should expect the attitude of the body to be such as to relax the involved muscle and particular movements which would stretch the muscle would be absent or limited. Such muscle injuries would probably recover fairly rapidly and completely.

Hematomas.—Extravasations and accumulations of blood in the muscles and other soft tissues must be expected from both direct and indirect trauma. Reference to this is made in other parts of this paper and needs no other mention here.

Traumatic Arthritis and Synovitis.—Traumatic arthritis of the vertebral and sacro-iliac joints is important from a medico-legal viewpoint, and a lengthy article might be devoted to this subject alone. Anatomically speaking, these joints are of such a nature as to be affected by both acute and chronic (mild repeated) trauma, as we know occurs in other regions. Due to the enormous leverage applied, the average person is constantly subjecting his lumbar vertebrae to hundreds and the athlete or laborer to thousands of pounds of pressure. It is not surprising, therefore, that this region frequently shows arthritic changes. Kummell's disease is an extreme type of lesion believed to be of traumatic origin. The great difficulty would

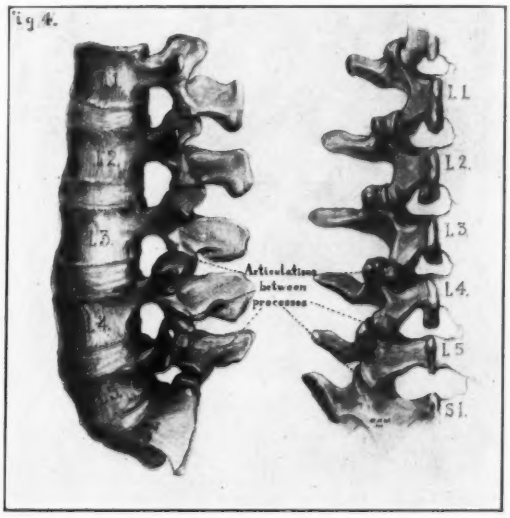

Fig. 4

Lateral and posterior view of the lumbar vertebrae showing especially the joints between the articular processes and their relations to the intervertebral openings. These relationships explain how chronic arthritis of these joints and sprains of the adjacent ligaments may produce nerve root irritation and pressure.

seem to be in differentiating between a purely traumatic arthritis and infectious or metabolic arthritis aggravated by injury, often a very mild injury. We believe this can usually be done with an accurate history and a thorough examination.

Compression and Contusion of Spinal Cord and Nerves.—The anatomy of the spine in relation to cord injury has already been briefly discussed. We repeat that such injury rarely occurs without vertebral fracture or dislocation, or both. The cord proper does not extend below the level of the first lumbar vertebrae and is well protected in the spinal canal in the thoracic region. The cervical region is not being considered in this paper. The cauda equina may be injured in the same way as the cord above, but is less liable to a complete compression or severance.

Injuries to the cord and its cauda within the spinal canal are better understood than the milder injuries involving the spinal nerves at and near their exits through the inter-vertebral foramina. The cases presenting clinical evidence of nerve root and peripheral nerve pressure and irritation, without demonstrable fractures or dislocations, are numerous and confusing. We have been especially interested in trying to determine the anatomical and pathological explanation of these manifestations. This is obviously a difficult problem, otherwise we should not now be seeking its solution. You are all familiar with the patient who, after either an apparently mild or severe trauma, has pain in the lower back with referred pains in the gluteal or sciatic nerve regions, or both, often associated with a persistent list of the body (scoliosis) toward the opposite side and more or less "flat back" and limitation of lumbar movements. This, we believe, represents the effects of pressure or irritation of the nerves, usually the fourth or fifth lumbar, at their exits, through the inter-vertebral foramina and surrounding ligaments. There are several factors which we believe important in this connection. We have already mentioned fractures of the articular processes, which may not be visible in even the best radiograms, and ruptures of ligaments about these processes. Osteo-arthritis of the spine is often present in patients past thirty-five or forty years of age, and if we remember that there is an articulation forming the anterior boundary and another the posterior boundary of each inter-vertebral foramen, and that arthritic joints are easily damaged by even mild strains, it is easy to picture a resulting swelling and infiltration in the surrounding soft tissues which will cause nerve pressure and irritation similar to that due to ligament rupture or fracture. The attitude of the patient above mentioned is explained by the involuntary effort to assume and maintain the posture which increases the size of the foramen and soft tissue spaces about the affected nerve and thus relieve pressure as much as possible. It is interesting to observe that the foramina between the fifth lumbar and first sacral vertebrae are smaller than those above, though the fifth lumbar nerves are larger than any of the others. Moreover, these spaces are smaller and the nerves more crowded in individuals having skeletons of the broad, thin type of vertebrae and in those with massive transverse processes, such as the sacralized fifth lumbar vertebrae. It is, therefore, to be expected that such skeletal types are more subject to backache, with and without referred pains, than those with more space for the accommodation of the roots of the lower lumbar nerves. It has been claimed by many that sciatic and other referred pains in the lower extremities are often due to strains and subluxations of the sacro-iliac joints. We cannot find any anatomical or other sound basis for this assumption.

In conclusion, we wish to thank Dr. F. H. Swett, of the Department of Anatomy, and Miss Susan Wilkes, Staff Artist, for their valuable assistance in the preparation of this paper and the accompanying illustrations.

*Discussion follows paper of Dr. Martin, page 650.

ANOMALIES OF THE SPINAL COLUMN AND PELVIS*

By J. M. MARTIN, M.D.,
Dallas, Texas.

This subject was suggested to me by your Chairman for a radiological consideration. I recognize my responsibility to both the injured and the injurer when I undertake the task of clearing up conditions that have on numerous occasions been a bone of contention in many a hard-fought legal battle. The subject of anomalies of the spine and pelvis is of the greatest clinical interest to the surgeon and constitutes an immense economic problem to be met by every employer of labor. The radiograph has made possible a detailed study of the bones of

*Read in Section on Railway Surgery (Southern States Association of Railway Surgeons), Southern Medical Association, Twenty-Second Annual Meeting. Asheville, North Carolina, November 12-15, 1928.

the human spine and pelvis both in health and disease. The errors in development and the congenital defects of the bony structures are usually classed under the heading of anomalies.

During a radiographic examination many of these anomalies are found by accident, are symptomless, and are of little importance except when discovered in connection with an examination of an injury, real or pretended. At such times a differential diagnosis between an anomaly and an actual injury is of first importance. Undoubtedly, judgment for damages has frequently been assessed against corporations because of bony variations in the spinal column due to defects in structural formation instead of actual injury. Because of this fact, all institutions employing labor in hazardous occupations should have employes' spines x-rayed before they are allowed to go to work. In this way many symptomless bony defects would be found and recorded and thousands of dollars in judgments and court costs could be saved annually.

At the meeting of the American Medical Association, last Spring at Minneapolis, Dr. Bohart, of Chicago, read an exhaustive paper on this subject. He had examined 931 applicants for positions with a railroad, who passed perfect physical examinations and were without symptoms referable to the spine. A large number of anomalies were found when these cases were x-rayed. Spurs and lipping of the vertebrae were common and when these were added to the anomalies approximately 5 per cent showed spinal defects. With the exception of the arthritic cases, those having abnormalities were able to do heavy work such as is required of railroad firemen and switchmen without suffering discomfort. While a large number of individuals under ordinary conditions are able to do the hardest kind of work in spite of one or more spinal defects, it is but reasonable to suppose that deformed vertebrae do weaken the back and lay it liable to over-strain or other injuries that a perfectly formed back would withstand without injury of any kind. Those who complain of weak backs and lower back pain after heavy work may show demonstrable bony anomalies, but often do not.

Hypertrophic changes in the vertebrae cannot be classed under the head of anomalies, but they are of such frequent occurrence as to call for a careful consideration. These conditions frequently cause no symptoms, but following an injury they may be the cause of pain and discomfort of long standing. Their origin is doubtful. They have been considered the result of an infection or of long, continued excessive strain. No attempt will be made to exhaust the subject of the spinal and pelvic regions, but a few of the frequent and most important variations from the normal will be mentioned.

When we are asked the number of spinal vertebrae, we unhesitatingly say twenty-four, and promptly divide them into seven cervical, twelve dorsal, and five lumbar. Occasionally there are eight cervical and rarely but six. The second and third cervical are sometimes completely fused. The transverse processes may be very long and broad or small and short. Cervical ribs, either rudimentary or complete, are often encountered on one or both sides, but often produce no symptoms.

In the dorsal region interesting anomalies are frequently seen. There may be a segment of an extra vertebrae bearing an extra rib in one side. The extra rib may be incomplete and unite with the rib above or below it. In rare instances the rib is complete. Asymmetrical development of one or more vertebrae usually causes distortion of the spinal column by bending it out of its normal alignment. In one of our cases, however, an extra vertebral segment in the dorsal region seemed to cause no change whatever in the alignment of the column. The facets that receive the heads of the ribs are generally regularly placed either wholly on a vertebral body or with halves on adjoining portions of two vertebral bodies. This arrangement is frequently changed, the facets being entirely on the vertebral bodies, whereas they should be situated across the interspaces. Such an arrangement causes an uneven spacing of the ribs on one side and has been used as evidence of fractured or dislocated ribs. It is often a very difficult matter successfully to demonstrate these anomalies to attending physicians, because radiographs of this region are likely to be confusing to those of us who have had considerable experience in their interpretation.

The lumbar spine may consist of six vertebrae and rarely there are but four. The fifth is occasionally completely fused with the sacrum and the transverse processes are sacralized, giving the appearance of a somewhat elongated sacrum. The transverse processes are variable and may be almost any size from mere rudimentary outgrowths to long and broad projections that resemble rudimentary ribs. In rare cases, one or more transverse processes fail to unite with the vertebrae, giving the appearance of a fracture. This anomaly usually occurs in the process of

the first lumbar vertebrae. At any point in the spinal column there may be malformation of the pedicles and laminae, to the extent sometimes that the neural arches are incomplete. The laminae may fail to unite or they may pass each other and fail to form a spinous process. It is regrettable but true, that the radiograph cannot always be made to show evidence of injuries and anomalies in the pedicles and laminae. In spina bifida radiographic evidence is dependable, even when the deformity is slight.

Variations are not uncommon in the fifth lumbar vertebrae. Dwight has said that "Anomalies of the fifth lumbar are so common that we hardly know what the normal should be." As in the dorsal region, a segment of a vertebrae may be encountered on one side. This has occurred once in our experience. The fifth vertebrae is wedge-shaped and is the cause of a sharp angulation with a lateral scoliosis. The antero-posterior radiograph of the lumbo-sacral spine may show one or both of the transverse processes of the fifth lumbar reaching down to the sacrum or overlying the crest of the ilium. Unless stereoscopic plates are made it will be impossible to determine whether there is a bony or cartilaginous union between the transverse process and the sacrum or ilium.

Spondylolisthesis, though usually associated with an injury, should be classed under the head of anomalies, because it rarely occurs in an anatomically normal back. The forward tilting of the sacrum and anomalies in the articulations of the sacrum and fifth lumbar, together with stretching of the ligaments and muscles of this region under strain, may allow the fifth lumbar to slip forward on the sacrum. The change is often slight at first and may be unilateral and progress slowly without revealing the true condition and the diagnosis may not be made for months after the injury.

In radiographing the spinal column multiple positions should be used, including an antero-posterior, lateral and oblique views. Sometimes a lateral should be made from each side. Only recently in making a lateral radiograph of the lumbar region of an injured spine a roughening was evident on the anterior margin of the fourth vertebrae. Because the man had been x-rayed from the left side before, he was turned over and an exposure was made from the right side. When the plate was developed I was surprised to find a distinct fracture of the body of the fourth vertebrae, which was not clear in the radiograph made from the other side.

Eternal vigilance is the price of success in this class or work, and strive as we may, conditions frequently come up that are difficult of solution.

720 Medical Arts Building.

DISCUSSION (*Abstract*)

Papers of Drs. Billington and Cunningham and Dr. Martin.

Dr. A. E. Chace, Texarkana, Ark.—Repeated trauma is not the question involved, but one particular trauma at one particular time.

Dr. J. R. Garner, Atlanta, Ga.—Reference has been made to the work being done by Dr. W. H. Bohart and also by Dr. D. B. Moss, in Chicago, in taking x-rays of men applying for positions. Their records will show us something in the future because their x-rays are of men complaining of absolutely nothing and denying having any physical disqualifications. They apply for positions thinking they are absolutely normal. This work is being done to establish the true condition and observe the ultimate outcome.

About thirteen years ago I treated a man in Atlanta and had an x-ray made of his lower spine and pelvis which showed a so-called occult spina bifida. He disappeared and later was found suing a railroad in Peoria, Illinois. He was kept in a hospital in Chicago for a period of twelve months. A diagnosis of fracture was made, and the case went to trial. I went to Peoria, taking the x-ray I had made fifteen years before. He still had exactly the same condition, and the case was won by the railroad. He is at present as well as I am. When he found he had no money coming to him he recovered. The work that is being done in Chicago will be of great benefit to the railroads throughout the Country sooner or later. By tracing back records of applicants we may gain helpful information.

I am very glad Dr. Billington referred to the displacement of the sacro-iliac as being practically impossible without some fracture of the rim of the pelvis. I do not think you can dislocate the sacro-iliac joint without a fracture somewhere around the sacrum or without a dislocation in the symphysis pubis at the same time.

Dr. Martin mentioned the possibility of a turning of one side of the fifth lumbar vertebrae on the sacrum which might pass for some time without detection. I had such a case about five years ago. The man had so many other injuries that this particular one was overlooked. Several ribs were fractured close to the spinal column, and many other things were wrong with him. He could not be turned in order to take a lateral view and the antero-posterior view did not show the injury in the lumbo-sacral region. After walking on an artificial leg he found he could not support his weight on the back. Later a lateral x-ray picture showed very distinctly that the last lumbar vertebrae had slipped on one side and the real trouble was apparent.

In dislocations of the fifth lumbar vertebrae on the sacrum, you will find many times that several x-rays will have to be taken to obtain a satisfactory picture, but if the antero-posterior picture shows an increased density at that locality finally a lateral picture will show it. The very marked increase in density has

a cause, and it is generally the slipping forward of the body of the fifth lumbar vertebrae.

Dr. Billington (closing).—The question of the importance of these definite hypertrophic changes has been discussed and there seems to be a little difference of opinion as to their cause, whether or not they are pathologic conditions. I think they are unquestionably pathologic and I think we should consider two causative factors, chronic infection and trauma. Working people are constantly being subjected to trauma, but it is also true that many of them have bad teeth or other focal infections that are damaging and irritating those joint structures, and constant irritation, so far as bone is concerned, is always accompanied by hypertrophic change. It is Nature's effort to throw out tissue to combat the irritant.

I will mention two problems which I think are of special interest to railroad companies: first, accurate diagnosis of genuine disability and of malingering; and, second, avoidance of employment of men with arthritic spines which will be easily injured.

If you know how to observe those cases and know what the movements mean and what they should be to protect that particular region, you can differentiate between genuine disability and voluntary muscle resistance. If the patient has no involuntary rigidity of his spine, no consistently guarded movements, no changes in the contour, no faulty posture or flattening of the back, then there is not much the matter with that spine. Now if the railroads will take up this problem of examining the backs of their employes when they apply for a position and make a record as to whether that man's spine is freely movable within normal limits for a man of his age, and see whether he has any limitation of motion, any change in the normal curves or in the posture, that, together with the x-ray record, should be very valuable.

Dr. Martin (closing).—One anomaly that I did not mention is an ununited transverse process in the lumbar region. Two such cases have been observed and they were found while radiographing the patients for other conditions than injuries. Had these patients been injured, I might have thought the anomalies were actual fractures. These cases have taught me a lesson that will make me very cautious in the future when reporting fractures of transverse processes of the lumbar spine. This anomaly is usually found on one side of the first lumbar vertebrae.

If I employed labor in hazardous occupations, I should wish radiographic records of each individual before he entered my service; and I should probably include the entire bony structure in this examination. Dr. Bohart made the statement that when arthritic conditions were included, about 50 per cent of the cases examined showed abnormalities of the spine. It astounded me to learn that there is such a large percentage of spinal abnormalities among men who are able to work without complaint on a railroad as firemen, switchmen, or engineers.

It is often a difficult matter to make good radiographs of actual injuries. My surgical friends have often said to me: "Why don't you show a good plate of the part that is injured when other parts of the spine are perfectly clear?" Owing to an extravasation of blood into the tissues surrounding the injury and other tissue changes, the density of the region is often increased and the radiograph is lacking in detail exactly where we most desire clearness. However, if radiographs are made at different angles, with the proper factors, results should be satisfactory in most cases.

BASIC PRINCIPLES IN THE PUBLIC HEALTH FIELD*

By JOHN A. FERRELL, M.D., DR.P.H.,†
New York, N. Y.

The topic assigned to me by your Program Committee embraces certain subjects about which the leaders in public health are not in complete agreement. Presumably I am expected to present my individual views as to a few of the important principles in public health as a basis for discussion. An exhaustive treatment of the subject would not be practicable on this occasion.

Facts, scientifically demonstrated, should serve as the proper basis for conducting public health procedures. Such facts fortunately are now available to a degree sufficient to permit rational practices on a broad scale applicable to most problems now generally accepted as falling within the field of public health. To conduct public health activities without regard to established knowledge would involve unnecessary speculation with money, health and life, and to say the least, would be almost certainly wasteful. The field worker in routine activities, then, should devote himself mainly to the application of available knowledge. He can adhere to this principle and find more to do than he and all the recruits who can be enlisted for decades can complete. The procedure under consideration may be the formulation, enactment or enforcement of law. It may be the immunization of whole communities against disease, the protection of water and food supplies, the ventilation of houses, or the selection and preparation of food. The method employed may involve the enforcement of laws and regulations or education by means of demonstration. In all cases the procedure, whatever it may be, should be based upon established knowledge.

Although the present store of knowledge as to public health is large and the justifiable range of routine activities is wide, there are a number of important diseases which are not yet well enough understood to give the health worker an effective method of procedure. Influenza, infantile paralysis, measles and cancer would fall in this group. Moreover, there are a number of diseases which have disappeared from large population groups where present knowledge has

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†Rockefeller Foundation.

been applied under favorable conditions, but their complete eradication has not been attained and may not be until new and more complete knowledge is available and until more direct and economical methods of contol are discovered or developed. Malaria, yellow fever, tuberculosis and venereal diseases, as examples, could be placed in this group. It is imperative, then, to carry on laboratory and field investigations with a view to supplying the knowledge, the scientific facts that are lacking as a basis for efficient, economical and rational procedures in the control of disease and in the conservation of health. Workers in this aspect of the public health field should have an exceptionally thorough scientific background. They should be so trained and so situated that they can pursue their observations closely and check them from every practicable angle. Fortunately the growth of interest in this aspect of public health is rivaling the application of existing knowledge, and both are contributing toward making public health a distinct and attractive professional field.

Particular emphasis should be placed upon the importance of making service in the public health field a worth-while vocation. The character of public health work will be but a reflection of the personnel that labors in this field. To be efficient, the worker must have special training, each for his particular task. But training alone will not suffice. To attain a commanding status, the health service must draw to its ranks the best type of citizen: one who possesses character, personality, knowledge, judgment, industry, poise and tact. One having these qualifications can succeed in any one of a number of professions. If public health is to attract him, it must offer a dignified status, employment for his full time, with compensation and security of tenure upon an efficiency basis and not upon one of political activity or favor. The field of public health is so broad and so varied as to the types of workers required that the subject of training personnel will be discussed presently. We may say here with reference to the conditions of service, including appointment, compensation and tenure, that more satisfactory conditions as a rule have prevailed where matters of appointment and policies are vested in boards whose members have fairly long and overlapping terms than where they are completely controlled by the incoming governor, mayor or administration. Of course, the public as well as the politicians must be educated to understand that the spoils system of politics and efficiency in measures for protecting health and saving life are incompatible. The realization of this concept will require wise leadership in educational effort and, too, we must realize that the time element will be a factor. A really phenomenal change for the better in public opinion has been observable during the past fifteen or twenty years and the outlook is indeed encouraging.

Public health work is in the main a function of government. There should be coordination and correlation of policy and effort among Federal, state and local, that is, city or county, health services. These branches of government have cooperated with satisfactory results in many parts of the Country under permissive legal and financial agreements. The autonomy of no branch of government need be sacrificed when the cooperation is in the nature of a partnership rather than a mandatory arrangement. Adherence to this principle does not, however, preclude non-governmental health agencies from supporting and strengthening sound governmental programs and from otherwise promoting the cause of public health. For example, private funds in many instances have permitted the establishment of new and fruitful services for which public funds were not available until after the value and importance of the work had been demonstrated. Moreover, as a general rule, it is difficult to secure or to use public funds for experimental work, yet everyone knows of the profound influence which certain discoveries have exerted in controlling disease. The absence today of many of the scourges of the past is directly attributable to the discovery of new knowledge and to the development of new methods. There is, then, a distinct opportunity for service for the private health agency.

The scope of the public health field is extremely broad. Its boundaries are not yet clearly defined. They vary in different parts of the Country. It is not necessary at this time to determine their exact location. If we tried, we should succeed only in starting a controversy. In every part of the Country at present the field of public health is sufficiently broad to permit the utilization of all the money and personnel that will be available for years to come. The epidemiology and control of preventable disease, vital statistics, laboratories, sanitation and personal hygiene are divisions of the public health field. Each of these major branches in turn includes a number of specialties. Communicable diseases, for example, require teaching and often practical work by diagnosticians, bacteriologists, immunologists, chemists, pathologists, physiologists, biologists,

entomologists, zoologists, and others. Likewise the laboratory service requires personnel equipped to make bacteriological, biological and chemical tests in connection with the diagnosis of disease, testing for susceptibility to disease, in producing immunity and in combating disease after its onset has occurred. The same condition with reference to the necessity for special knowledge will hold to a greater or less degree for the other basic subjects that are embraced in the public health field.

The carrying of the available knowledge to the individual, the home, the school and the business establishment, so that it will be applied in the most extensive and practical manner likewise is one of the most important responsibilities of the public health service. The number of workers required for this phase of the health service must be relatively very large. They must be trained in making human contacts. Their results are most satisfactory when the desired end is attained with a minimum of law enforcement and with a maximum of educational effort that leads to cheerful cooperation. Training and experience in field methods are just as essential as are highly technical laboratory methods. Obviously the highly trained workers cannot be secured in sufficient numbers and given the additional training for field service that the situation requires. Experience has taught that, although situations frequently arise which render it necessary for the technical expert to share in the field activities, the practical application of all the public health knowledge that the various branches of science have placed at our disposal can be entrusted to three classes of field workers: (1) the medical health officer; (2) the public health nurse; and (3) the sanitary inspector.

The medical health officer, in addition to having general supervision of the work of the group as a whole, should perform those duties for which his medical and public health training fits him and for which the training of the nurse and inspector are inadequate. The handling of cases of contagious diseases, the diagnosing of diseases and defects, the application of necessary measures of prophylaxis, dealing with the medical profession, and numerous other activities naturally should be performed by the medical health officer or at least under his immediate supervision. The nurse in her visits to the home and to the school is, by nature and training, ideally fitted to deal with mothers and children and to teach and apply much of the scientific knowledge now available. She is an indispensable factor in the public health scheme and numerically she now exceeds any other class of public health workers and the field for her usefulness is growing rapidly.

The sanitary inspector, too, is indispensable. He should devote his efforts to safeguarding the human environment. Water and food supplies must be protected. Dangerous water must be purified. Human wastes must be disposed of in a sanitary manner. The breeding of disease-carrying insects must be controlled. Nuisances must be abated. The trained inspector can perform service of this kind effectively and perhaps more satisfactorily than could the physician and certainly at a smaller cost. The health machinery also requires stenographers, bookkeepers and clerks. When one considers the breadth of the public health field and the varied classes of workers required for its conduct and the training they must have, each for his particular share of the work, it will be recognized that the recruiting and training of personnel is a big problem and that it would not be wise to attempt, certainly at the present stage of development, to establish for all health workers a uniform minimum eligibility requirement. If too low a minimum training requirement for all workers should be established, it might be abused and result in positive harm. Perhaps the workers could be classified into a few important groups and minimum training standards established for each group. For the specialists or highly technical workers (such as bacteriologists, epidemiologists, statisticians and sanitary engineers), the schools of public health, the colleges and universities, in consultation with the health authorities, can set up minimum training requirements.

The demand for practical field workers so far exceeds the supply that the public health administrators have been unable to wait for the schools to produce them. Undoubtedly the situation has improved and will continue to do so. In the past, the health officer as a rule had to be a qualified physician, either licensed in the state where he was to do health work or eligible for license. The nurse had to be a graduate and usually licensed. The requirements for the sanitary inspector were less definite. During the past four or five years the trend has been to require that all new employes for field service, usually in the county or similar organizations, either have previous successful experience in public health work or take practical intensive field training at approved or satisfactory training stations for periods of from four to twelve weeks.

Stations of this character have been operated in Alabama, Ohio, Mississippi, Tennessee and Missouri, and have been aided by the organization with which I am connected. A station in the sense here used is merely a county health organization of the standard type, which has a health officer, nurse and inspector who are exceptionally well qualified and who are interested in and willing to devote time and effort to practical teaching and demonstration. As far as practical the time they spend in teaching is offset by giving the county superior talent which its unsupplemented budget would not command and also in certain instances by supplying additional personnel. Other well-conducted health departments can be and have been used for providing similar training. This type of training serves a useful purpose just now. It is merely a temporary and transitional practice school. It may be expected in time to be improved and to be as closely allied with schools of public health as it now is with health departments. In its logical development it may function as university extension work. This might aid the university to assist in recruiting personnel to meet the present emergency and to attract from the ranks of health workers students who should utilize the available facilities for more fundamental preparation for public health service.

With the growth in public health work as indicated by increased expenditures, by increased numbers of workers employed, by more secure and satisfying living conditions, by the disappearance of certain diseases which were once epidemic, by the prolongation of life and the reduction in death rates, the tendency of enthusiasts in the public health field is to start new activities before their value can be thoroughly tested by qualified investigators and to expand existing activities, some of which have not had their value tested with a due degree of thoroughness. Many of the more conservative leaders, however, feel very keenly the responsibility of spending large amounts of public funds. They think the possibilities of evaluating the various activities should be studied by competent persons. A beginning in this direction has been made. The task has not been found to be easy because basic data have not been collected in a way nor to a degree sufficient to warrant sweeping conclusions. Moreover, over a number of years so many variables operate as to limit the value of observation. Although the solution of the problem is not yet in sight, the situation is encouraging in that need for study and facts is recognized and already a number of workers are directing their energies in this direction.

SUMMARY

(1) Practices in public health should be based upon facts scientifically demonstrated.

(2) Investigation in the field and laboratory as a means of enlarging present knowledge is definitely indicated.

(3) Public health service should be a distinct professional field, requiring suitable preparation and insuring appointment, tenure, compensation and other conditions of service on an efficiency basis.

(4) Public health work is in the main a function of government, Federal, state and local, each autonomous but working in cooperation.

(5) There is a useful field for service for the unofficial health agency. It can strengthen, support and complement the official agency. It can engage in pioneering measures and in research where public funds are not available.

(6) The scope of public health, though not definitely defined as to its outer boundaries, is extremely broad and, as generally accepted, embraces many branches of science; and its conduct requires many workers of various types.

(7) The attracting, training, compensating, appraising and holding of the necessary personnel are pressing problems. The instruction ranges from short practical training in the field, in the nature of a short apprenticeship under favorable conditions, to the most thorough university courses. The demand for qualified personnel continues to exceed the supply.

(8) For the usual routine field activities, the health officer, nurse and inspector have proved indispensable.

(9) A method for the accurate evaluation of public health activities to avoid waste is greatly needed. Its development presents a difficult problem which, to a large degree, still awaits a solution.

DISCUSSION (*Abstract*)

Dr. W. F. Draper, Washington, D. C.—Public health authorities have demonstrated what cooperation can really be. They understand each other perfectly with a minimum of waste of effort and time. The relationships with the states and local agencies are most harmonious and satisfactory. The relationship between public health officials and practising physicians can be just as harmonious and helpful. Time and again I have received the cooperation and active support of the practising physicians in a county, without which I could not possibly have secured the establishment of that county health department.

The more the public health worker knows, the greater chance of service he has; however, I have seen splendid work done by public health officers who have had little special public health training. Dr. Ferrell has demonstrated that in an emergency officers can be turned out for county health work after a brief but intensive course in its essentials. In the flood states they have been rendering good service and we hope a large number of those county health workers will become members of permanent health units.

A study should be made of each public health activity to determine its importance in the whole program. Perhaps we are doing some things that are not necessary, and not doing some things we should. We could in such a study also examine the public health practices in foreign countries and, as a result, we should probably be able to evaluate and improve our present methods.

Dr. Frederick Taylor, High Point, N. C.—I was particularly interested in one point in Dr. Ferrell's paper: the fact that certain diseases have been almost impossible to deal with from a public health standpoint.

North Carolina is conducting this year an experiment to try to reduce the chronic degenerative diseases, the real wholesale killers: heart disease, kidney disease and cancer. As these diseases give no definitely recognizable symptoms, the one solution to the problem we have at present is to catch them before they give symptoms, if possible; to give as much attention to our bodies as we do to our banks and automobiles: have them examined periodically.

We are urging upon the profession of the State the annual examination of apparently healthy persons; we are urging the people to go to their physicians for this examination annually. The problem is too large to be dealt with by the public health men alone, or the insurance companies alone, though they have been pioneers in teaching us the value of this work. I believe the latest statistics show that the annual mortality in an annually examined group is somewhere between 18 and 24 per cent lower than the annual mortality of unexamined. How much is due to the examinations or to the fact that the more intelligent group takes better care of itself in other ways we do not know, but we have inaugurated this work because we believe it worth while. To work out this campaign we have a threefold method of approach: first, to arouse the medical profession to its value. Ofttimes, the practising physician has not been so aroused.

We give demonstrations in clinics. We like to examine the physicians themselves. The other day we found in 271 examinations of apparently healthy individuals 911 defects.

We have talks to various public groups: parent-teacher associations, county council meetings, women's clubs, civic clubs, radio. The death rate from chronic heart disease in our State is now double that from tuberculosis.

A massed cooperative movement between the medical profession and the public for the purpose of bringing about widespread periodic examinations of the people by their own physicians is destined to be the next great progressive move in preventive medicine, and in the saving of human life wholesale.

Dr. R. M'G. Carruth, New Roads, La.—In New Roads, we expect in the near future to inaugurate a campaign advocating annual or semi-annual examinations, and I have suggested to my younger confreres that it would be a good thing for us to begin on each other, to compare notes as to methods. With many physicians it is a question of looking at the tongue, feeling the pulse, taking the temperature and blood pressure, percussing and auscultating the chest, and saying "I think you are all right. You do not need any further examination."

My particular interest in making the examinations more thorough has been stimulated by my experience in trying to conform to the spirit of the present state hygienic marriage law in Louisiana. It has too often been my sad experience in examining those contemplating marriage, even those apparently in perfect health who conscientiously believe there is nothing wrong with them, to find a latent gonorrhea or a positive Wassermann. Examinations properly made will do much to bring to light these cases.

Dr. Roy K. Flannagan, Richmond, Va.—There is always danger of health officers becoming side-tracked by interesting but relatively unimportant aspects of health work and failing to give due emphasis and consistent application to the fundamental things Dr. Ferrell has outlined. Let us be careful to keep our feet on the ground.

I confess to be tremendously interested in the periodic medical examination of apparently healthy individuals. This work offers great promise for the future, but the medical profession must develop the idea, set the pace and show health departments where they can with propriety help out. Until this is done health departments may well keep hands off.

The real meat of sound public health practice has been brought out by Dr. Ferrell, the basic things, such as epidemiology, vital statistics, laboratory diagnosis and sanitation. Let us know how to administer these well and then we can begin to ramify and perhaps be definitely concerned about those things which are now more or less in the stage of experimentation.

SEDIMENTATION RATE OF ERYTHROCYTES*†

By FRANCIS B. JOHNSON, M.D.,
Charleston, S. C.

Many of the older writers noticed, in the process of blood-letting, that blood cells settled much more rapidly in certain diseases than in the normal. However, with the giving up of this method of treatment, the point was forgotten.

This phenomenon was again brought to our attention in 1918 by Fahreaus,[1] who introduced it as a test for pregnancy, and showed that the rate was also accelerated in acute infections and malignancy. Many others have contributed to our knowledge of this phenomenon, so that we have now a very extensive literature on the subject, both from a clinical and technical standpoint.

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*Read in Section on Pathology, Southern Medical Association, Twenty-Second Annual Meeting, Asheville, North Carolina, November 12-15, 1928.

†From the Laboratory of Clinical Pathology, Medical College of the State of South Carolina.

TABLE 1

METHODS OF DETERMINING SEDIMENTATION RATE

Method	Blood Column Height	Blood Column Diameter	Blood Dilution	Amount of Diluted Blood	Anti-coagulant	
Fahraeus (1)	170 mm.	9.0 mm.	1-4	16 c. c.	Sod. Cit.	2%
Westergreen (2)	200 "	2.5 "	1-4	1 "	" "	3.7%
Linzenmeir (3)	50 "	5.0 "	1-4	1 "	" "	5.0%
Morriss (4)	200 "	2.+ "	1-4	1 "	" "	3.8%
Rubin (5)	200 "	2.+ "	No	1 "	Hirudin	
Cooper (6)	50 "	15.0 "	No	5 "	Pot. Oxal.	20%
Hunt (7)	200 "	2.+ "	1-4	1 "	Sod. Cit.	1.6%
Zeckner & Goodall (8)	73 "	15.0 "	1-4	10 "	" "	3.0%
Cutler (9) special pipette		2.5 "			" "	3.0%
Plass & Rourke (10)	100 "	3.8 "	No	1.1 "	Heparin	

Since the technic of Fahreaus was described, there have been introduced some fifteen modifications of technic and different methods of recording the results. This tends to make the interpretation of the test very confusing to the average clinician. It is appreciated that all methods are introduced with the object of simplifying the process and making more accurate the checking up of results. I disavow any attempt in this paper of introducing a new method. My object is to bring to your attention certain principles, in comparison with some of the methods already introduced, to promote better correlation of results. We cannot expect the general practitioner or surgeon to be familiar with the interpretation of all methods described. If certain general principles are understood the interpretation becomes much clearer.

In *Table 1* are demonstrated certain variations in some ten of the methods introduced. One sees that the methods differ as to height of column of blood, diameter of the column, the amount of blood used, the dilution or concentration, and the anti-coagulant used.

There are some five different methods of recording the results, the two most widely used being that of Westergreen and of Linzenmeir. In the former the number of millimeters of height of plasma above cells, is recorded in a definite time of one and two hours. Normally this is 1-7 mm. for the first hour. The modifications of this method include, recording the height of cell column, recording the volume per cent of plasma of cells, and graphic records. There is also included the determination of the cell colume at the end of 24 hours, or after centrifuging, from which certain valuable data may be obtained. In the method of Linzenmeir the time it takes the cells to settle a given distance, 18 mm., is determined. Normally this takes over 160 minutes. The settling of cells is much faster in women than in men, probably because of the lower proportion of cells and hemoglobin found.

There have been introduced methods of comparing the drop in one or two hours to the complete fall after standing twenty-four hours, or after centrifuging. Plass and Rourke[10] have introduced a method of taking the percentage of plasma at the end of one hour, divided by the percentage of plasma after centrifuging, times one hundred, giving the sedimentation rate in per cent. This gives a normal average for man of 11.6 per cent and women 30.9 per cent. Heparin is used as an anti-coagulant. This obviates some of the difficulties of the methods now in common use.

EXPERIMENTAL INVESTIGATIONS

My investigations have been to determine the influence of the following points on the rate or speed of sedimentation.

(1) The influence of height of the column of blood.

(2) The influence of the diameter of the column of blood.

(3) The influence of dilution and the ratio of cell volume to plasma volume.

(4) The influence of change of volume of blood.

(5) The effect of various dilutions of blood.

(6) The effect of various anti-coagulants.

(7) The effect of change of temperature at which the test is made.

(8) Comparison of sedimentation rate (Plass and Rourke[10]) to length of tube.

The results shown in the following tables are typical examples of many repeated tests. In most cases a rapid rate is shown as the effect was more decided.

TABLE 2

EFFECT OF HEIGHT OF COLUMN ON SEDIMENTATION

Diameter of All Tubes 5 mm.

Blood Column Height	Minutes to fall 18 mm.	Millimeters fall in One hour	Millimeters fall in Two hours
260 mm.	29 min.	53 mm.	98 mm.
200 "	34 "	50 "	78 "
100 "	35 "	30 "	47 "
65 "	38 "	25 "	38 "
50 "	45 "	21 "	24 "
25 "	.... "	15 "	16 "

In *Table 2* are shown the results of repeated tests using varying heights of column of cells in tubes of the same diameter. Sodium citrate 3.8 per cent was used as anti-coagulant and diluent. The results are given in the number of minutes it takes the cell column to drop 18 mm. and the fall in millimeters read at the end of one and two hours. It will be observed that the taller the column of blood the shorter time it takes to fall 18 mm., also the greater the number of millimeters fall in one and two hours. Therefore, it is important that the height of the column be the same to obtain comparable results.

TABLE 3

EFFECT OF DIAMETER OF BLOOD COLUMN ON SEDIMENTATION RATE

Height of Column of Blood in All Tubes 100 mm.

Blood Column Diameter	Minutes to fall 18 mm.	Millimeters fall in One hour	Millimeters fall in Two hours
2.5 mm.	30 min.	33. mm.	47 mm.
3.0 "	31 "	32. "	47 "
3.8 "	32 "	32. "	44 "
5.0 "	33 "	30. "	43 "
6.6 "	35 "	30. "	43 "
15.0 "	42 "	24. "	35 "

With tubes in which the columns of blood were of the same height and the diameters of the tubes were different, as shown in *Table 3*, there was very little difference noted in the rate of fall, with the exception of the centrifuge tube in which the diameter was 15 mm. at the top and tapered down. So this factor of diameter of column of blood has little to do with rate of fall.

A comparison of the combined effect of changing the height and diameter of the column of blood is shown in *Table 4*. Here the results check up fairly well on the previous findings in the previous tables. It is seen that the effect

TABLE 4

COMBINED EFFECT OF CHANGE IN HEIGHT AND DIAMETER OF COLUMN OF BLOOD ON SEDIMENTATION

Blood Column Height	Blood Column Diameter	Minutes to Fall 18 mm.	Millimeters Fall in One hour	Millimeters Fall in Two hours
260 mm.	2.5 mm.	20 min.	54 mm.	100 mm.
200 "	2.5 "	25 "	50 "	80 "
200 "	3.8 "	28 "	46 "	79 "
100 "	5.0 "	31 "	33 "	48 "
50 "	5.0 "	37 "	24 "	29 "
50 "	15.0 "	....	13 "	....

is mainly from change of height of column and not diameter, except where diameter is markedly increased.

The dilution of the blood being used for the test is an important factor and should be carefully made. The usual proportion is one part of diluting anti-coagulant to four parts of blood. This must be promptly added. Otherwise, if coagulation has even partly commenced, the test is interfered with.

This dilution in itself hastens the settling of the cells, and although the whole blood may be diluted 1-4, where there is a natural variation from normal in the proportion of plasma to cells, the actual dilution of plasma will be affected in consequence. In anemia, where we have an increase in proportion of plasma to cell volume, this is a very important point to remember.

TABLE 5

THE EFFECT OF ANEMIA OR VARIATION OF CELL VOLUME TO PLASMA VOLUME ON SEDIMENTATION RATE

Dare Hemoglobin Per Cent.	Minutes to fall 18 mm.	Millimeters fall in One hour.	Millimeters fall in Two hours.
84 %	180 Min.	5 mm.	8 mm.
75 %	175 "	12 "	16 "
62 %	70 "	20 "	22 "
38 %	27 "	29 "	32 "
20 %	17 "	40 "	42 "
11 %	13 "	46 "	47 "

Blood Column Height 50 mm., Diameter 5 mm.

In *Table 5* is shown the effect of change of cell volume to plasma volume by the addition of plasma to cells which were not packed by centrifuging, therefore, having considerable amount of plasma with them. We have not actually one volume of pure cells, but a volume diluted to give certain variations in the percentage of hemoglobin. Here is seen what a decided effect anemia will have on the rate of sedimentation. For approximately each 10 per cent fall in hemoglobin, the sedimentation rate increased from 50 to 100 per cent with more marked anemia.

TABLE 6

COMPARATIVE EFFECT OF ANTI-COAGULANTS ON SEDIMENTATION RATE

Diameter of all tubes 5 mm.
Height of column of blood 100 mm.

Anti-Coagulant	Dilution	Minutes to fall 18 mm.	Millimeters fall in one hour	Millimeters fall in two hours
Pot. Oxalate	No	21 min.	45 mm.	55 mm.
Heparin	No	25 "	45 "	54 "
Sod. Cit. 5%	1-4	35 "	34 "	48 "
" " 3.8%	1-4	36 "	32 "	47 "
" " 3%	1-4	36 "	31 "	45 "
" " 2.5%	1-4	30 "	30 "	45 "

Of the anti-coagulants used, sodium citrate is most widely adopted, the strength of solution varying from 5 to 1.6 per cent. Potassium oxalate, sodium oxalate, hirudin, and heparin are also used, but these are used in the dry form so that no dilution of the blood takes place. With tubes of the same diameter and height of column of blood, some of the various anti-coagulants were tried as demonstrated in *Table 6*. Here it will be noted that the speed of sedimentation was a little more rapid in the tubes with oxalate or heparin, than in those various dilutions of sodium citrate. The variation between the different solutions of citrate was only slight.

TABLE 7

EFFECT OF CHANGE OF TEMPERATURE ON RATE OF SEDIMENTATION

Diameter of All Tubes 3.8 mm.
Height of Column of Blood 100 mm.

Temp. C.	Minutes to fall 18 mm.	Millimeters fall in One hour	Millimeters fall in Two hours
6°	89 Min.	11 mm.	22 mm.
11°	88 "	13 "	24 "
20°	60 "	18 "	30 "
37°	52 "	23 "	35 "

The effect of variation of temperature at which the test is carried out is demonstrated in *Table 7*. The blood was placed in waterbaths, in incubator, in ice boxes, and at room temperature to get it at the right temperature before starting the test. The cells were allowed to settle at these temperatures. The table shows that the temperature has much to do with the speed at which the cells fall. The higher the temperature the more rapid the fall.

An effort was made to see if the method of Plass and Rourke would correlate some of the above factors in regard to the length and diameter of blood column. It will be seen from *Table 8* that in tubes of the same length, but of varying diameter, the rate was practically the same, but different in those tubes of varying length of column of cells. The method has one advantage in that it does make allowance for change of cell volume to plasma volume, naturally found in anemia, and the other methods do not.

TABLE 8

DETERMINATION OF SEDIMENTATION RATE (PLASS AND ROURKE)

Length of Blood Column	Diameter of Tube	Plasma After One Hour	Plasma After Cent. 20 Min. At 2500 Rev.	Sedimentation Rate
200 mm.	5.0 mm.	13.8%	65.4%	21.0%
100 "	5.0 "	22.0%	63.4%	34.9%
50 "	5.0 "	29.8%	64.0%	46.2%
200 "	3.8 "	13.4%	64.5%	20.4%
100 "	3.8 "	22.6%	66.0%	33.8%
50 "	3.8%	20.6%	65.9%	44.9%
200 "	2.5%	15.1%	65.4%	23.0%
100 "	2.5%	23.8%	66.8%	35.5%
50 "	2.5%	31.6%	64.8%	48.7%

Sedimentation rate equals: Plasma per cent after one hour X 100. Plasma per cent after cent. 20 min. at 2500 rev. per min.

DISCUSSION

That the lack of any one standard method of carrying out this test has led to a certain amount of confusion in regard to the interpretation of results is, I think, shown. There is, however, a broad margin left in what we may consider normal, and the rate in abnormal cases may fall to an extensive degree. Hence, minor differences can be overlooked if we bear in mind certain general principles that have been demonstrated: these are, the proper dilution of blood, the prevention of clotting, the comparative dimensions of the tubes in regard to height and diameter, the proper temperature of the blood when the test is carried out, and the importance of existing anemia.

The diameter of the tube within reasonable figures has little to do with the speed of fall of erythrocytes, while the length of column of blood has considerable effect. The longer the column the more rapid the rate. That a change in the temperature will have a decided effect of varying very much the speed of settling is also shown. This confirms the results of Gordon and Cohn.[11] It is therefore advisable that the test always be carried out at a stated temperature. A temperature of 20°-23° c. is recommended. This is a rather hard temperature to maintain in Southern climates during hot weather, but can best be done in a water bath. It is important to remember the effect of anemia as shown. By the use of the method of Plass and Rourke,[10] al-

lowance can be made for this effect of change of plasma volume to cell volume.

CLINICAL VALUE

The increased rate may be said to occur in all conditions where the higher products of protein destruction are found in the blood, by some said to be due to increased fibrinogen. The test is of particular value to the surgeon in inflammatory pelvic conditions of the female, and as a diagnostic and prognostic aid in tuberculosis. We must bear in mind the fact that the rate is increased in many other conditions, such as malignancy and pregnancy; in all febrile conditions; in many inflammatory conditions without fever; after the use of x-ray and radio-active substances therapeutically; in conditions showing high blood nitrogen retention, in cardiac decompensation, in benign tumors where destruction and resorption of cell products are occurring, as well as in malignant tumors; active syphilis; and for at least eight days after operations of any kind.

Over-enthusiasm occurring at first in regard to the value of this test has now given way to the realization of its restricted value.

As an indicator of inflammation, this test appears to be of more value in some cases than the total and differential blood count. As Polak[12] states, "The test never lies." There are, however, many things that may confuse it. It is more constantly present, but does not show as early an appearance as a leucocyte increase, for in acute appendicitis twenty-four hours may elapse before the sedimentation shows any decided increase in rate.

In the determination of the time or feasibility of operation in pelvic inflammatory conditions, the test undoubtedly is of greatest value if used repeatedly. In tuberculosis it may be of diagnostic value, in that pulmonary tuberculosis is said not to exist actively unless the rate is increased. However, its chief value here is in prognosis.

In the diagnosis of pregnancy it is of little value because in the first place the increased rate occurs only after the third month, and in the second place such confusing tumors as fibroids will show an increased rate when degeneration is taking place in the tumor.

CONCLUSIONS

(1) The introduction of many modifications of the original method of determining the sedimentation rate of erythrocytes has caused considerable confusion in interpreting results by different workers.

(2) Efforts to correlate these methods have been made by the writer, but have met with little success due to certain factors: the length of tube column of blood, the dilution of blood, the anti-coagulant used, the temperature at which the test is carried out, and existing anemia, all have an important effect on the rate and should be taken into consideration.

(3) The method of Plass and Rourke avoids some of the difficulties in regard to the variations in cell volume to plasma volume which would be found in anemia, and offers the method of choice, if we could all decide to adopt one method.

(4) The test is one of limited value. It is of most value to the gynecologist in indicating the favorable time for surgical interference in pelvic inflammatory conditions, especially when the temperature, blood count, and clinical observations fail. This test cannot, and should not, replace clinical methods or the blood count.*

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DISCUSSION (*Abstract*)

Dr. Johnson (closing).—I said in the beginning of the paper that I disavowed introducing any new method. There are already some fifteen methods for doing this test, and I felt that if I tried to bring up a new method everybody would get up and walk out.

The value of this test is limited, but if we recognize its limits it is of some aid and should be used, taking into consideration varying conditions showing an increased rate of sedimentation.

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MASSIVE COLLAPSE OF THE LUNG: REPORT OF AN ADDITIONAL CASE*†

By CHARLES T. STONE, M.D., F.A.C.P., Galveston, Tex.

Sir John Rose Bradford[1] has ably defined massive collapse of the lung as

"an unusual condition in which the lung without the presence of any gross lesion such as bronchial obstruction, pleural effusion, etc., interfering with the free entry of air, becomes airless to a greater or less degree, and, therefore, useless for respiratory purposes."

It is a well known fact to clinicians and pathologists alike that smaller or larger areas of pulmonary atelectasis may result from or be associated with many diseases of the respiratory system, e. g., pneumonia, both lobar and lobular, tuberculosis, pneumothorax, pleural effusion, tumors of the lungs, and so forth. It has also been amply demonstrated in Chevalier Jackson's Bronchoscopic Clinic that foreign bodies in the bronchi may be followed by a collapse of a considerable amount of lung tissue. However, it is not these types due to disease of the respiratory system, or to foreign body bronchial obstruction, that we wish to emphasize at this time, but rather the type of massive atelectatic collapse included in Bradford's definition given above.

William Pasteur,[2] of London, was the first to point out clearly, in 1890, the existence of massive collapse of the lung, as he found it occurring in seven cases of post-diphtheritic paralysis of the diaphragm. In all fairness, however, creditable mention should be made of the work and observations of earlier writers. It appears that as early as 1844 two Frenchmen, Legendre and Bailly,[3] recognized the condition in children. In 1852, the elder Meigs[4] reported clinical cases of *atelectasis pulmonum* in infants, and in 1879 Meigs,[5] the younger, made further similar observations. Lichtheim,[6] in 1879, published a striking account of the production of pulmonary atelectasis in rabbits by ligation of the main bronchus, but it remained for Pasteur to place the condition on a firm clinical foundation. He continued his studies of massive collapse, and in 1908[7] further amplified his views about it. In 1914[8] he showed that the condition often occurred as a post-operative complication, most frequently following abdominal operations. Sir John Rose Bradford,[9] during the World War, made numerous observations of the condition in soldiers who had received chest injuries. Since the publications of Pasteur and Bradford, a considerable literature has accumulated upon the subject, and yet it appears that the profession has been slow to accept and recognize the importance of the condition.

Incidence.—Massive collapse of the lung is doubtless of far more frequent occurrence than is generally believed because of the ease with which it may be confused with other diseases of the chest, especially lobar pneumonia. Pasteur[8] found that it occurred 16 times in 2,000 laparotomies which he studied, and Bradford[10] estimated that it was a complication in 5-10 per cent of all chest injuries in military practice, while Mastics, Spittler and McNamee[11] concluded that, based on their observations, atelectasis comprised approximately 70 per cent of all post-operative pulmonary complications. If the above figures are even remotely correct, large numbers of cases are going begging for a correct diagnosis.

In February, 1928, Coryllos and Birnbaum[12] reviewed the literature and collected a total of 119 cases, and mentioned 50 more that had previously been reported by Mastics, Spittler and McNamee.[11] Since their report I have been able to gather an additional 76 cases from all sources, which, together with the case I am herewith presenting, make a grand total of 246 cases. In *Table I* are listed the relevant data from the 76 cases not included in the previous report.

My own attention was first directed to this subject by the late Dr. J. E. Thompson, Professor of Surgery in the University of Texas, in 1917, through an article of Bradford's[10] published in a War Monograph by the British Government. Since that time diligent search among the post-operative cases seen in consultation with the Surgical Service of the John Sealy Hospital has produced but one typical case of massive collapse of the lung, the case history of which will be detailed later.

Etiology.—In military practice wounds of the chest, severe or trivial, penetrating or non-penetrating, furnish the larger number of cases; while in civil medicine abdominal operations, especially appendectomy, hernia repair, gynecological procedures, operations on the thyroid gland, hemorrhoidectomy, and fractures of the pelvis and femur are most often responsible. Aside from the traumatic and post-operative

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group, massive collapse of the lung may complicate such diseases as pneumonia, tuberculosis, poliomyelitis, meningitis, pericarditis with effusion, and so forth. In the post-operative cases it has more often occurred when ether has been used as the anesthetic, but it may follow the use of any general anesthetic, and has even been reported after local or spinal anesthesia.

Pathogenesis.—With regard to the actual mechanism by which the collapse is produced, much uncertainty exists. In any event it appears necessary that a bronchial obstruction of some nature precede the collapse. This is quite possibly due to a sudden reflex closure of the finer bronchioles, either by spasm or swelling of the mucous membrane, which traps the air in the vesicles and from which it is subsequently absorbed, with resulting atelectasis. The various predisposing factors which appear to favor the development of the lesion may be listed as follows:

(1) The posture of the patient, especially when lying upon one side for long periods, and not using the abdominal muscles in respiration, such as occurs post-operatively.

(2) Diaphragmatic inhibition.

(3) Diaphragmatic paralysis.

(4) Bronchial mucus or other secretion which might produce obstruction to the smaller bronchi.

(5) Paralysis of the muscles of respiration exclusive of the diaphragm. Experimentally, the condition cannot be produced by anything except mechanical bronchial obstruction. Respiratory muscle paralysis and phrenicotomy have never resulted in massive collapse. The explanation which seems the best that can be offered for this complex phenomenon is that of Sante:[13]

"Some infection or insult to the region of the vagus supply produces a reflex action on the bronchioles, permitting their temporary collapse. Once approximated, the walls of these bronchioles are held in apposition by cohesion, collapse of the lung rapidly following, owing to the absorption of alveolar air by the circulating blood."

Scott,[14] however, while accepting the belief that closure of the smaller bronchi is the most probable immediate cause of the pulmonary collapse, advances the hypothesis of Hoover that the closure of the tubes is the result of bronchial occlusion induced by swelling of the mucosa, which is of reflex origin, and which is analogous to angio-neurotic edema. He further states that the causative factor affects both lungs, but in unequal degree. In support of this claim he asserts that during the height of massive collapse the vital capacity may be reduced to 25 per cent of normal, a figure much lower than would obtain if only one lung were affected. Whatever the final solution to the problem may be, it seems safe for the present to assert that the evidence favors a reflex nervous factor as essential for the production of clinical massive collapse.

Varieties.—Taking into consideration the exciting etiological factors, it was suggested by Leopold[15] that massive collapse of the lung might be classified as follows:

(1) Post-operative.

(2) Traumatic.

(3) That which follows complete obstruction of the air passages.

(4) That which occurs during the course of post-diphtheritic multiple paralysis.

(5) Postural.

(6) That which occurs during the course of certain infections, particularly pneumonia and typhoid fever.

Such a grouping of cases is of distinct value from the standpoint of statistical study, and in securing more accurate data upon the various types of massive collapse.

Morbid Anatomy.—Since so few uncomplicated cases end fatally, there has been but scant opportunity afforded for study of the morbid anatomy. It occurs at any age, and though with much greater frequency among males, in the reported cases, it is found in both sexes. In the 76 cases presented in this report the right lung was the seat of the process 47 times, the left lung 24 times, and both lungs five times (*Table I*). It may affect part or all of one lobe, more than one lobe, or the whole of one lung. In the event of bilateral involvement of the entire pulmonary tissue sudden death is inevitable, as in a case reported by Bergamini and Shepard. In the pathological descriptions of Bradford[1] the lung affected has been heavy, shrunken, bluish in color, airless, and often intensely congested. No evidence of mechanical bronchial obstruction has been observed except in the foreign body cases, and there the mechanism is probably very different from that in the non-foreign body cases. In massive collapse of the lung there is an enormous increase in the negative intrapleural pressure on the involved side, and this causes an elevation of the diaphragm, retraction of the chest wall, and a drawing of the heart and mediastinal structures toward the side of the collapse. In a case reported by Sante[13] the retracted chest wall promptly expanded to normal when the thorax was opened at autopsy. The associated extreme

pulmonary engorgement is obviously due to the increase in negative pressure about the lung, and possibly to an accompanying vaso-dilation, producing the condition referred to by Leopold[16] as "drowned lung."

The time of onset after the provocative cause is usually within the first 48 hours. Rarely it occurs during the course of the abdominal operation, and in some cases it may be deferred for several days (*Table I*).

Symptoms.—The symptoms are very variable, and there may be none, even with the whole of one lung completely collapsed. Such cases would naturally escape detection unless careful examination of the chest was made. In the majority of instances, however, when massive collapse occurs there is a dyspnea of greater or less severity, at times extremely urgent and distressing. Pain over the chest is of frequent occurrence. Cough of varying intensity is generally present, although it may be absent entirely. At first dry, the cough is apt to become productive of a mucoid or muco-purulent sputum, but the expectoration of bloody sputum has infrequently been observed except in the traumatic cases. With the onset of collapse there is, as a rule, a sharp rise in the temperature up to 101 to 104° F. during the height of the symptoms. A moderate or slight cyanosis is often present. A leucocytosis, due to an increase in the number of polymorphonuclear neutrophils, of from 12,000 to 20,000 is the rule in the reported cases in which blood counts have been given (*Table I*). This finding still further misleads the physician to suspect that pneumonia is the cause of the symptoms.

Physical Signs.—The physical signs of massive collapse are quite characteristic. The affected side of the chest is retracted, smaller than the normal, and immobile. The interspaces are narrowed. The chest is absolutely dull or flat on percussion. Transmission of the spoken or whispered voice varies, and may be either diminished, increased, or absent. Likewise the breath sounds may be quite different in different patients, or at different times in the same patient. With increased fremitus the breath sounds are bronchial, or amphoric, and bronchophony and pectoriloquy are present, while with absent or diminished fremitus the breath sounds are also diminished or absent. In any event, the most characteristic physical sign is the drawing of the heart toward the affected side. The diaphragm is elevated, and this may be ascertained by percussion on the left side, but much less readily on the right, owing to the position of the liver.

X-Ray Findings.—The x-ray shows the collapsed lung, or portion of the lung so affected, to be perfectly opaque. There are also seen elevation of the diaphragm, displacement of the heart and mediastinal contents, especially the trachea, toward the affected side, and the ribs lie closer together than upon the sound side. Sante,[13] in commenting upon the dense opacity of the lung in massive collapse, contrasts the picture with the relatively transparent lung of pneumothorax, and adds that the difference has never been adequately explained. It would appear that the dense shadow of the lung in massive collapse is due to the engorgement and edema of the lung, which is absent in pneumothorax, either artificial or spontaneous.

Diagnosis.—With such findings, and particularly since the physical signs and x-ray shadow strongly resemble those of pulmonary consolidation, it is not remarkable that we have long been content to designate the condition as "postoperative pneumonia." However, if the predisposing causes are considered, the onset symptoms and physical signs carefully noted, and especial care is given to observe the displacement of the heart toward the lesion, one can scarcely confuse this significant picture with that of any other disease.

Prognosis.—As to prognosis, the outlook is good unless the condition which the massive collapse complicates leads to an unfavorable termination, or unless, as occasionally happens, the collapse involves all of both lungs. So far as the massive collapse itself is concerned, one may reasonably expect a return to normal within one to three weeks. Complications are usually absent during convalescence, but there have been mentioned purulent bronchitis, pleurisy, lobular pneumonia, and pulmonary edema.

Treatment.—The treatment of massive collapse of the lung is first of all that of the primary disease. As to the collapsed lung itself, the patient should be encouraged to breathe deeply, and to move about upon the sound side. In one instance Sante[17] observed, under the fluoroscope, the rapid re-inflation of a collapsed lung by rolling the patient upon the unaffected side of the chest and exciting the cough reflex, but this treatment is not always successful. Bronchoscopic removal of mucus from the bronchi has been advocated by Jackson and his associates, but this method is sharply limited in its usefulness and appears to be of doubtful value

unless, of course, a foreign body is present. It seems likely that the coughing incident to bronchoscopy is of greater significance than the removal of the mucus. Artificial pneumothorax has been used by Farris[18] on the theory that the symptoms may, in part, be the result of cardiac displacement, which can be overcome by creating a positive intrapleural pressure on the affected side. Morphia, in appropriate doses, during the period of urgent symptoms, is indicated. Otherwise the treatment is entirely symptomatic and varies with the condition preceding the massive collapse. Scott[14] studied the effect of atropin and epinephrin in massive collapse because of the action of these drugs upon the vagus and sympathetic nerves, respectively, but saw no change in the pulmonary condition following their administration to the point of full physiological effect.

The report of the following case illustrates perfectly the characteristics of the condition:

REPORT OF A CASE

C. H., an American school boy, 13 years of age, was admitted to the surgical service of Dr. A. O. Singleton (to whom I am indebted for this report) in the John Sealy Hospital at 7:00 p. m. February 24, 1928, complaining of pain in the right side of the abdomen. His illness had begun during sleep the night previous to admission into the Hospital. He had retired feeling well, but was aroused from sleep with cramp-like pains all over the abdomen. Later, the pain became localized in the lower part of the abdomen, on the right side. He indicated the location of the pain over McBurney's point. There were also present nausea, vomiting, and headache. He gave no history of similar preceding attacks. Cough, expectoration, and other respiratory symptoms were absent.

Physical examination showed a boy slightly underweight, who was alert, intelligent and cooperative. Apparently his pain was not extremely severe. The temperature was 101° F., the pulse was 100 per minute, and of good volume. There was increased muscle tension over the right lower half of the abdomen and rather severe pain on pressure over McBurney's point. Otherwise the physical examination was negative. The urinalysis showed: amber color, acid reaction, specific gravity 1.035, a heavy trace of albumin, sugar negative, a slight flocculent sediment, and microscopically, a few leucocytes, some in clumps, a few hyaline and granular casts, and an occasional epithelial cell. The white and differential blood count was: leucocytes 9,200, polymorphonuclear neutrophils 73 per cent, lymphocytes 20 per cent, large mononuclears 2 per cent, and transitionals 5 per cent. A diagnosis of acute appendicitis was made, and immediate operation was decided upon. At 8:10 p. m. (February 24, 1928), under ether anesthesia, the abdomen was opened through a McBurney incision, and an acutely inflamed appendix filled with pus was removed. There was no soiling of the peritoneum and the wound was closed without drainage. The operation was completed in twenty-five minutes, during which time the pulse ranged between 98 and 104 per minute, and the respirations were 23 to 25 per minute.

The following day the patient felt well except for pain in the right side of the abdomen at the site of the incision and inability to void, which necessitated catheterization. The morning of the second day was uneventful and he was considered to be having a normal convalescence. The temperature was 100° F., pulse 90, respiration 24. At 3:00 of the second day, 43 hours after operation, there was a sudden rise of temperature to 104.4° F., associated with very intense dyspnea and slight cyanosis. The interne found the patient in great respiratory distress. The respirations were shallow, very rapid, 50 per minute, and he appeared in grave danger. The pulse rate was 120 per minute. The physical signs over the chest were limitation of movement on the right side, with dullness on percussion, and an absence of breath sounds over the entire right lung. The patient lay upon the right side and resented any effort to turn him. Believing that this was a case of lobar pneumonia, a cool sponge bath was given and pneumococcus antigen 1 c. c. was administered subcutaneously. The next morning a medical consultation was held and the following findings were recorded: the left chest was expanded more than normal. The respiratory rate was greatly increased (36 per minute). The right chest was distinctly smaller than the left. Palpation showed the diminished respiratory excursion on the right and markedly increased tactile fremitus. The right chest was flat from apex to base on percussion. The left lung was hyperresonant throughout. By auscultation over the right chest the breath sounds in the upper part down to the level of the fourth rib were bronchial; below this area the sounds were practically absent. No rales were heard. On the left side the breath sounds were those of exaggerated vesicular breathing. Transmission of whispered and spoken voice sounds on the left was normal, while on the right it was greatly increased.

The left border of cardiac dullness was 4 cm. from the mid-sternal line in the fourth intercostal space. The right border of cardiac dullness, as nearly as could be made out in view of the dullness of the right chest, was 6 cm. at the level of the second rib. The cardiac impulse could be felt in both the right and left fourth interspaces in each instance about 3 cm. from the mid-sternal line. The heart sounds were heard more distinctly to the right of the sternum and were not abnormal. A diagnosis of massive collapse of the right lung was made, and an x-ray of the chest was ordered.

The x-ray showed a dense opacity on the right side of the chest extending from diaphragm to apex. The heart was drawn to the right side and the right thorax was considerably collapsed.

Roentgenologic diagnosis was massive collapse of the right lung.

Following the suggestion of Sante,[17] the patient was rolled about on the sound side in an effort to bring about rapid re-inflation of the collapsed lung, but without success. He was then returned to the ward, where his course was carefully followed from day to day, both by physical examination and by x-ray.

The change in the physical signs were as follows: February 28, 1928 (36 hours after the development of the collapse), the upper part of the right chest down to the fourth rib level was tympanitic on percussion and the breathing was amphoric in quality over the same area. The other signs were the same as those obtained February 27.

February 29, 1928 (60 hours after the onset of the collapse), there was some return of the respiratory excursion in the upper part of the right chest, but the

TABLE I. FINDINGS IN SEVENTY-SIX REPORTED CASES OF MASSIVE COLLAPSE OF THE LUNG *

Case No.	AUTHOR AND SOURCE	Age	Sex	Condition Preceding Massive Collapse	Anesthetic	Time of Onset	Temp.	Pulse	Resp.	Cyanosis	Dyspnoea	Pain	Cough	Sputum	White Blood Cells	Lung or Part of Lung Involved	Result	Duration
1	Ball, R. P. Arch. Path. 5:763, 1928	30	F	Thyroidectomy		24 hrs.	103.6	146	+	+						L.L.L.	Death	24 hrs.
2	Ball, R. P. Arch. Path. 5:763, 1928	17	M	Thyroidectomy, partial	Local NO_2 O_2	6 hrs.	104	160	30	+						R.L.L.	Death	12-18 hrs.
3	Ball, R. P. Arch. Path. 5:763, 1928	52	F	Thyroidectomy		5 days				+	+					L.L.L. R.L.L. R.M.L.	Death	18 hrs.
4	Ball, R. P. Arch. Path. 5:763, 1928	49	F	Thyroidectomy						+	+					Left	Death	12 hrs.
5	Sante, L. R. Am.J.Roentgenol. 20:213,1928	19	M	Asthmatic Attacks			103.2			+	+					Left	Rec.	48 hrs.
6	Riebel, I. F. M.J.&Record 128:321, 1928	18	M	Herniotomy (bilat)	NO_2 Ether	4 days	102.6	104	42	+	+	+				L.L.L.	Rec.	24 hrs.
7	Riebel, I. F. M.J.&Record 128:321, 1928		M	Fracture Ribs		24 hrs.	99	120	40	+						Right	Rec.	23 days
8	Scott,W.J.M. & Joelson,J.J.F. Arch. Surg. 15:855, 1927	24	M	Pyelotomy-Left	NO_2 Ether	24 hrs.	103	140	40	+						Right	Rec.	3 weeks
				Pyelotomy-Right	NO_2 Ether	48 hrs.	104.6	120	40	+						Left	Rec.	19 days
9	Scott,W.J.M. & Joelson,J.J.F. Arch. Surg. 15:855, 1927	28	M	Pyelotomy. L. Cystotomy	NO_2 Ether	24 hrs.	+	+	+							R.M.&L.L.	Rec.	11-16 days
10	Scott,W.J.M. & Joelson,J.J.F. Arch. Surg. 15:855, 1927	26	M	Fusion Sacro-Iliac Joint R		7 days						+	+			Left	Rec.	
11	Hahn, L. J. Internat.J. Med.&Surg. 37:232, 1924			Nephrectomy R		2 days										Left	Rec.	
12	Jackson, C. & Lee, W. E. Ann. Surg. 82:364, 1925	12	M	Appendiceal Abscess		3 days	101.3	140	52							Right	Rec.	
13	Scott, W.J.M. & Cutler, E.C. J.A.M.A. 90: 1759, 1928		M	Ligation Superior Thyroid Vessels	NO_2	24 hrs.										Left	Death	3 days
14	Scott,W.J.M. & Cutler,E.C. J.A.M.A. 90: 1759, 1928			Cholecystectomy	Ether	24 hrs.										R.&L.L.L.	Rec.	
15	Smith,F.J. & Davidson,E.C. J.Michigan M.Soc.26:295,1927	24	M	Herniotomy, L	Ether	24 hrs.	100.6	124	40			+	+		20,100	Right	Rec.	6 days
16	Smith,F.J. & Davidson,E.C. J.Michigan M.Soc.26:295,1927	26	M	Herniotomy (bilat)	Ether	24-48 hrs.						+	+	+	5,500	Left	Rec.	7 days
17	Smith,F.J. & Davidson,E.C. J.Michigan M.Soc.26:295,1927	25	M	Herniotomy Ventral Hernia Repair	Ethylene	12 hrs.	101.8	90	46	+					20,500	R.L.L.	Rec.	4 days
18	Smith,F.J. & Davidson,E.C. J.Michigan M.Soc.26:295,1927	30	M	Herniotomy (bilat)	Ether & Ethylene	24 hrs.	103	160	40	+			+		19,700	R.L.L.	Rec.	12 days
19	Smith,F.J. & Davidson,E.C. J.Michigan M.Soc.26:295,1927	42	M	Herniotomy	Ether	24 hrs.	102	100	48	+			+		16,000	R.U.&M.L.	Rec.	8 days
20	Smith,F.J. & Davidson,E.C. J.Michigan M.Soc.26:295,1927	35	M	Herniotomy (bilat)	Ethylene	24 hrs.	102	116	28						18,850	R.L.L.	Rec.	6 days
21	Smith,F.J. & Davidson,E.C. J.Michigan M.Soc.26:295,1927	41	M	Herniotomy (bilat)	Ether & Ethylene	24 hrs.	102.4	104	30	+			+		11,200	L.L.L.	Rec.	5 days
22	Farris, H. A. Canad. M.A.J. 15:808, 1925										+		+	+		Right	Rec.	6 weeks
23	Farris, H. A. Canad. M.A.J. 15:808, 1925	48	M	Separation Omental Adhesions		24 hrs.	102.2	140	50	+	+	+	+			Right	Rec.	8 days

24	Lund, C. C. & Ritvo, M. Bost.M.&S.J. 190:1103,1924	18	M	Appendicitis (gangrenous)	Ether	30 hrs.				+			+			Right	Rec.	15 days
25	Lund, C. C. & Ritvo, M. Bost.M.&S.J. 190:1103,1924	28	M	Herniotomy (bilat)	Ether	30 hrs.						+	+	+		Right	Rec.	20 days
26	Regan, J. C. Lancet, 2:1222, 1924	10 mo.	F	Acute Poliomyelitis		?			+	+	+		+		11,200	R.&L.L.L.	Rec.	20-30 days
27	Hirschboeck, F. J. Minnesota Med. 7:414, 1924		F	Salpingoophorectomy Double	Ether	36 hrs.	+	+	+		+		+			Right	Rec.	
28	Elwyn, H. Bost.M.&S.J. 190:1108, 1924	17	M	Lung Abscess		?					+		+			R.M.&L.L.	Rec.	
29	Churchill, E. D. Arch. Surg. 11:489, 1925	51	M	Herniotomy	Ether	36 hrs.					+		+	+		R.L.L.	Rec.	
30	Churchill, E. D. Arch. Surg. 11:489, 1925	28	M	Repair Ventral Hernia	Ether		+	+	+				+	+		Right	Rec.	
31	Churchill, E. D. Arch. Surg. 11:489, 1925	52	F	Fracture Spine Transverse Myelitis		Several Days					+		+			L.L.L.	Rec.	
32	Churchill, E. D. Arch. Surg. 11:489, 1925	63	M	Removal Dermoid Cyst Thigh	Ether	15 days					+	+	+			R.L.L.	Death	45 days
33	Churchill, E. D. Arch. Surg. 11:489, 1925	65	F	Neoplasm Left Hypochondrium												L.L.L.	Death	
34	Touhy, E. L.	48	M	Appendectomy Gastro-enterostomy	NO_2 O_2	12 hrs.	+	160	+		+					R.&L.L.L.	Death	
35	Peffard, T. A. Minnesota Med. 10:5, 1927	14	F	Pneumonia		10 days	+				+		+		15,000 22,000	Right	Rec.	5 days
36	Peffard, T. A. Minnesota Med. 10:5, 1927	52	M	Pneumonia		?	+	+	+		+				21,000		Rec.	20-30 days
37	Peffard, T. A. Minnesota Med. 10:5, 1927	60	F	Pneumonia—Right			+	+	+		+				12,000 30,000	Left	Death	
38	Peffard, T. A. Minnesota Med. 10:5, 1927	13	M	Broncho-pneumonia												R.U.L.	Rec.	
39	Keith,D.Y., Keith,J.P., & Bell, J. C. Kentucky M.J., 25:268, 1927	70	F	Cholecystectomy	??	10 days			30		+					R.L.L.	Rec.	
40	Keith, D.Y., Keith,J.P., & Bell, J. C. Kentucky M.J., 25:268, 1927	13	M	Appendicitis—Acute	??	3 days				+	+	+	+	+		Left	Rec.	5 days
41	Garrett, D. S. J. Okla., M.A. 25:268, 1927		M	Cholecystectomy Appendectomy		18 hrs.	104	118		+		+	+	+		Right	Rec.	4 days
42	Shellito, J. C. J. Iowa Med.Soc.16:186,1926	12	F	Appendicitis Gangrenous	Ether	36 hrs	+	+	+		+	+	+			R.L.L.	Rec.	10-12 days
43	Hunt, E. L. Boston, M.&S.J. 194:58,1926	21	M	Perforated Duodenal Ulcer	??	3 days	103	125	36	+		+				Right	Rec.	8-10 days
44	Hunt, E.L. Boston, M.&S.J. 194:58,1926	59	M	Acute Cholecystitis with Sub-Phrenic Abscess	Not Operated		+	+	+							R.L.L.	Rec.	
45	McKinney,J.J., Porter,W.B. South.M.J. 20:681, 1927	22	M	Repair Ventral Hernia	NO_2 Ether	24 hrs.	104	150	48							Right	Rec.	15 days
46	McKinney,J.J., Porter,W.B. South.M.J. 20:681, 1927	20	M	Gastro-enterostomy	NO_2 Ether	9 days	106.2	160	44							R.L.L.	Rec.	3 days
47	McKinney,J.J., Porter,W.B. South.M.J. 20:681, 1927	45	M	Colon Resection Carcinoma	Ether	24 hrs.	102.2	130	30							Right	Rec.	2 days
48	McKinney,J.J., Porter,W.B. South.M.J. 20:681, 1927	76	M	Prostatectomy Suprapub.	NO_2 Local	24 hrs.	103	130								Right	Death Uremia	15 days
49	McKinney,J.J., Porter,W.B. South.M.J. 20:681, 1927	34	F	Hysterectomy Supravag.		18 days		120	34	+	+					L.L.L.	Rec.	7 days
50	McKinney,J.J., Porter,W.B. South.M.J. 20:681, 1927	27	M	Cholecystectomy Appendectomy	Ether	72 hrs.	104	132	36							Right	Rec.	13 days
51	McKinney,J.J., Porter,W.B. South.M.J. 20:681, 1927	21	M	Perforated Duodenal Ulcer	NO_2 Ether	24 hrs.	102	140	40							Right	Rec.	11 days
52	Elkins, D. C. Ann. Surg. 86:885,1927	18	M	Herniotomy	Ether	24-48 hrs.	104	120	30							Right	Rec.	14-18 days
53	Elkins, D. C. Ann. Surg. 86:885,1927	44	M	Fracture Dislocation L. Humerus. Fracture L. Tibia		?	No Symptoms									L.L.L.		3 days

TABLE I. FINDINGS IN SEVENTY-SIX REPORTED CASES OF MASSIVE COLLAPSE OF THE LUNG*

Case No.	AUTHOR AND SOURCE	Age	Sex	Condition Preceding Massive Collapse	Anesthetic	Time of Onset	Temp.	Pulse	Resp.	Cyanosis	Dyspnoea	Pain	Cough	Sputum	White Blood Cells	Lung or Part of Lung Involved	Result	Duration
54	Elkins, D. C. Ann. Surg. 86:885,1927	39	F	Sarcoma Thigh												Left	?	?
55	Sante, L. R. Radiology 8:1, 1927	5	M	Appendicitis-Acute	Ether	33 days										R.U.L.	Death Septicemi	
56	Halperin, G. Surg.Gnec.Obst. 41:50, 1925	33	M	Cholecystectomy &Appendectomy	NO_2 Local Ether	48 hrs.	102.2	120	36		+		+			R.L.L.	Rec.	13 days
57	Stoloff, E. G. Am.J.Dis.Child. 35:239,1928	2	F	Early Tuberculosis		+	+	+			+		+			Left	Rec.	4 days
58	Asper, G. P. U.S.Vet.Beau.M.Bull. 3:1143,1927	32	M	Nephrectomy—L	Ether	30 hrs	103	140	35	+	+	+	+			Right	Rec.	30-60 days
59	Eads, M. L. Boston, M.&S.J.195:258,1926	30	F	Perineorrhaphy Ventrosuspension	NO_2 Ether	6 hrs	102.5	140	40	+	+					R.L.L.	Rec.	11 days
60	Trout, H.W. & Hayter,H.M. Virginia Med. Monthly 53:214, 1926	15	F	Appendicitis—Acute	Ethylene	24 hrs.	103.2	132	48						20,000	Right	Rec.	16 days
61	Trout,H.W. & Hayter,H.M. Virginia Med. Monthly 53:214, 1926	16	M	Appendicitis—Acute	Ethylene	54 hrs.	104	132	32	+		+			21,000	R.L.&M.L.	Rec.	3 days
62	Trout,H.W. & Hayter,H.M. Virginia Med. Monthly 53:214, 1926	16	M	Appendicitis—Gangrenous	Ethylene	12 hrs.	102.4	100	28	+		+	+		20,000	R.L.L.	Rec.	3 days
63	Trout,H.W. & Hayter,H.M. Virginia Med. Monthly 53:214, 1926	22	M	Appendicitis—Acute	Ethylene	72 hrs.	100.6	130	30	+		+			13,000	R.L.L.	Rec.	2 days
64	Beals, J. A. J. Florida M.A. 13:14, 1928	22	M	Herniotomy (bilat)		18 hrs.	104.4	140	40	+						Right	Rec.	2 weeks
65	Gwyn, N. B. Internat. Clin. 1:136, 1926	20	M	Appendicitis—Subacute	Ether	12 hrs.	+	+	+	+	+	+				Right	Rec.	17 days
66	Gwyn, N. B. Internat. Clin. 1:136, 1926		M	Appendicitis—Acute	Ether	24 hrs.	+	+	+	+			+	+		Left	Rec.	7 days
67	Gwyn, N. B. Internat. Clin. 1:136, 1926	26	M	Appendiceal Abscess	Ether	10 hrs.				+	+	+				Right	Rec.	10 days
68	Gwyn, N. B. Internat. Clin. 1:136, 1926		M	Bullet Wound Right Chest		45 hrs.										Left	Rec.	Few days
69	Solem, G. O. Med.Cln.N.Amer.11:105,1927	22	F	Tonsillectomy	General	10 days	99.8	90	28	+	+	+	+		21,000	Left	Rec.	18-24 days
70	Packard, E. W. Am. Rev. Tuberc. 18:7, 1928	25	F	Fibroid Tuberculosis Upper 1-3 Left Lung		Acute					+		+			L.L.L.		Permanent

71	Packard, E. W. Am. Rev. Tuberc. 18:7, 1928	30	F	Tuberculosis Upper 1-3 Right Lung & Upper 2-3 Left Lung	Acute	101			+	+	+			Left			3 years
72	Packard, E. W. Am. Rev. Tuberc. 18:7, 1928	30	F	Hilus Tuberculosis left Lung	Acute	102			+	+	+			Left			Permanent
73	Packard, E. W. Am. Rev. Tuberc. 18:7, 1928	44	M	Tuberculosis & Carcinoma Lung	Acute				+	+	+			Left			Permanent
74	Packard, E. W. Am. Rev. Tuberc. 18:7, 1928	52	M	Tumor Upper Lobe right Lung					+	+	+			Right			Permanent
75	Baer, J. L. Surg.Gynec.Obst. 37:97,1923	26	F	Cesarean Section Fibroid Broad Ligament	48 hrs.	101	168	+	+				23,000	Right	Rec.		2 weeks
76	Klein, Franz Med.Klin. 24:179,1928	73	M	Amputation Leg Arteriosclerotic Gangrene	30 days	99	+	+		+				Right	Rec.		1 day

* I am indebted to Dr. E. H. Schwab for valuable assistance in compiling data from the literature.

lower half remained practically immobile. Tactile fremitus was slightly increased over the right upper lobe, absent in the right base, and normal on the left side. By percussion the right lung was hyperresonant above the level of the fifth rib, and was dull from there down. The left lung was hyperresonant throughout. On auscultation the breath sounds over the right upper lobe were broncho-vesicular, right lower distant broncho-vesicular, and over the whole of the left lung they were slightly exaggerated vesicular. The transmission of the spoken voice, while showing much less difference on the two sides, was still increased in the right upper lobe and diminished in the right base. By that time the heart sounds were heard more clearly to the left of the sternum, but were still distinctly audible on the right side. The left border of cardiac dullness was 6 cm. from the mid-sternal line in the fifth intercostal space, and the right border of cardiac dullness was 4 cm. from the mid-sternal line in the fourth intercostal space.

From that time on the progress toward normal chest signs was rapid, so that by March 3, 1928, the respiratory system was again normal and the heart had returned to its pre-collapse position. On March 1, a few coarse, moist rales were heard diffusely scattered over both lungs. Thus, in six days complete recovery from the massive pulmonary collapse had taken place. During the whole illness, cough was never a severe symptom, the sputum was scant and mucoid, and at no time was blood present in it. The findings in this case leave no room for doubt as to the correct diagnosis: massive atelectatic collapse of the lung secondary to appendectomy.

CONCLUSIONS

(1) Massive collapse of the lung is a distinct disease entity which can be positively recognized both by the physical signs and the roentgen ray examination.

(2) It is frequently overlooked because of the ease with which it is confused with lobar pneumonia.

(3) The cause is not definitely known, but it is probably of reflex nervous origin.

(4) The salient facts about the condition have been presented; and,

(5) A typical case is reported.

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DISCUSSION (*Abstract*)

Dr. J. S. McLester, Birmingham, Ala.—Dr. Stone has told us that massive collapse of the lung occurs in the widest variety of conditions. It would appear that anything that interferes with the mechanism of respiration predisposes to this disease. It is hard for me to believe that massive collapse of the lung should come from a mere contraction or temporary closing of the smaller bronchi. It seems much more likely, as was suggested by Hoover, that a collection of mucus occurs in a bronchus, or perhaps a swelling of the mucous membrane, and that this interferes with the respiratory mechanism in such a manner as to lead to massive collapse.

Dr. John W. Scott, Lexington, Ky.—Though it is only in recent years that it has been generally recognized, massive collapse of the lung is by no means rare. I have met with three cases on my hospital service in the past year. It is, as the essayist has said, frequently overlooked. This is in large measure due to the fact that the possibility of such a thing is only beginning to dawn in the mind of many.

The plugging of a large division bronchus with mucus has been emphasized as the usual cause by many, particularly by Jackson. Others feel that the role of the diaphragm is important. A recent case seems to point to the latter. A negro vaudeville dancer attempting to do the "split" completed the act awkwardly and struck the floor with great violence. Severe pain in the abdomen and a degree of shock followed, though no definite visceral injury was recognized. Four days later massive collapse of the right lung occurred.

The other two cases followed abdominal operations of moderate severity. The onset of symptoms occurs more suddenly than in post-operative pneumonia. There is sudden chest pain and severe dyspnea. The pulse respiratory ratio is lowered even more than in pneumonia.

The pathognomonic physical sign is displacement of the mediastinum, which is recognizable both on physical examination and x-ray. It is hardly possible to fail to recognize the condition either on physical examination or x-ray if one has once seen it.

The patients I have seen recovered spontaneously in two or three days and since most of them have an abdominal wound I have felt reluctant to subject them to either bronchoscopy or the rolling maneuver of Sante.

Dr. Charles H. Cocke, Asheville, N. C.—I have reported a perfectly normal four day old baby who developed this condition, with classical signs as well as x-ray evidence, which cleared up completely within six days. Another case was one of suspected tuberculosis that proved to have a primary endobronchial malignancy, which diagnosis was confirmed by bronchoscope and eventually by autopsy.

I think this condition is overlooked much more often than has been supposed. Dr. Jackson and Dr. Lee have reproduced it in dogs, using mucilage of acacia.

In my case there was a complete displacement of the heart to the right chest, there being no signs of the heart on the left side in the second roentgenogram. We fortunately had the first picture showing that the heart was in normal position before the atelectasis occurred. There was also tracheal displacement. There is, too, the rather high diaphragm which lends some weight to the possibiity that something is wrong with the mechanism there. If you will stick a pneumothorax needle attached to the manometer into these cases, you will find some explanation for this enormous displacement in a very marked increase in the negative intrapleural pressure.

Dr. Stone (closing).—It seems likely from the information we have at present about massive collapse of the lung that there is more than one mechanism which accounts for the production of the condition in different subjects. There are probably numerous contributary factors. The influence of posture is, I am sure, quite important.

Dr. Webb, of Colorado Springs, treats his tuberculous patients by placing shot bags over the diseased area to bring about restricted respiratory movements on that side, and teaches them to lie upon the affected side. He showed some x-ray pictures at the meeting of the American College of Physicians last year that looked almost like the pictures that we get by x-ray in massive collapse of the lung. He simply had the patients lie on the involved side, thus restricting their respiratory movements.

Dr. Scott introduces a very valuable point in that we find this condition only as often as we look for it. As Pasteur himself said, in St. Bartholomew's, there was not a case found before 1914 and in the next several years he discovered sixteen cases in 2000 laparotomies.

CHRONIC URTICARIA, A THYRO-ADRENAL SYNDROME: HOW TO DETERMINE THE DIRECTION OF THE DYSFUNCTION, AND WHAT TYPE OF AGENTS TO EMPLOY IN ITS TREATMENT*

By J. N. Roussel, M.D.,
New Orleans, La.

In 1873 Tilbury Fox said (and he was wise in his generation) that

"The treatment of urticaria, especially chronic urticaria, is most unsatisfactorily laid down in books . . . . that it is one of the most difficult and unsatisfactory of all diseases to cure; and, in fact, the treatment of chronic urticaria is most tiresome and difficult."

Generally speaking, the reader is told to correct any digestive disorder, improve the general health, and use remedies to allay the itching, all of which is academic, and sounds well, but means nothing.

*Read in Section on Dermatology and Syphilology, Southern Medical Association, Twenty-Second Annual Meeting, Asheville, North Carolina, November 12-15, 1928.

Alexander Pope said that "It is with our judgments like our watches, none go just alike, but each believes his own."

It is my own judgment that of all the tormenting diseases that the Almighty has seen fit to impose upon us suffering mortals, chronic urticaria might safely be put very near the top of the list.

While this has been going on for countless ages, I cannot find where any comprehensive method of treatment has ever been proposed. By this I mean a method by which we are able to determine what type of drug to use in a given case.

It is doubtless true that practically everything in the pharmacopeia has been used by someone at some time, but always without any reasonably certain expectation of satisfactory results.

The treatment of chronic urticaria has caused more embarrassment to more men than that of the whole wart family. Those who have not been embarrassed by warts, at some time or other, are not numerous, I am sure. Chronic urticaria, warts and the heathen Chinee are, in the words of Bret Harte, "peculiar."

The difference between acute and chronic urticaria is one of degree, mainly, in the underlying pathology. That certain articles of food will induce an attack in either case, there is little doubt. Likewise will chemical agents, light, heat, and what not, do the same. But the mere statement that an individual has become sensitized to any agent, and that in order to effect a cure this agent must be avoided, probably forever, I think, is entirely out of line with the facts in the premises.

If an individual who has eaten certain articles of food and has been in contact with whatever agent may now be the apparent cause of his misery probably for thirty or forty years, suddenly finds himself unable to continue this contact further, then he is the victim of some serious metabolic disturbance, which should be determined and corrected before any good can be accomplished.

To tell a patient that he must avoid this, that, or the other thing, in order to obtain relief, is a therapeutic fallacy and a makeshift. That the cause should be removed is academic. There can be no argument against this proposition, but there is such a thing as putting the cart before the horse.

The real reason that one gets urticaria is not that he has eaten some shell fish which he has probably eaten daily for a generation or more. It is because something has gone definitely wrong with him. The shell fish is incidental, probably acting as a catalytic agent, initiating and changing the velocity and force of the vasomotor impulses.

Now what controls the vasomotor impulses?

In order to make clear the possibilities of a departure from the normal vasomotor control, a review of the mechanism of this control may be of assistance.

That every non-striated muscle fibre, and every organ in the body has a double nerve supply from the involuntary nervous apparatus, I think there is ample evidence. That irritation or stimulation of one branch of this system produces vaso-dilatation, and stimulation of the other branch causes vaso-constriction, there is little doubt.

The simplest and most common examples of vaso motor control are those of blushing and paling. A mild stimulus, such as a sense of embarrassment, causes a dilatation of the vessels of the face, with a resulting blushing, and a sharp stimulus, such as fright, produces vaso-constriction, or paling, instead.

While these are palpable facts there is little doubt that the basic control of the vasomotor apparatus lies in the endocrin system; and, while one part of this system may be as important as another, the thyroid and suprarenal divisions have attracted more attention than the others.

There appears to exist a close relationship between the thyroid and suprarenal glands, which has to do with the vaso-dilating and vaso-constricting mechanism of the involuntary nervous apparatus.

It is definitely known that the secretion of the thyroid gland is a vaso-dilator, when introduced into the system by any route, and it is also definitely known that at least one part of the secretion of the suprarenal gland, adrenalin, is a vaso-constrictor which, however, does not appear to operate as well by mouth as by the hypodermic or intravenous route.

It has been observed that a person with a complete absence of the thyroid shows no tendency to blushing or peripheral vaso-dilatation, but on the contrary is in a state of marked chronic peripheral vaso-constriction. On the other hand, it is a well known fact that the absence of the suprarenal glands induces a state

of great depression in which evidences of vasodilatation are apparent on all sides.

It is, therefore, quite evident that if the endocrinous "hook-up" (to use a radio expression) is complete, and the force and frequency of the vasomotor impulses has not been upset, we shall have no urticaria; and, it is with these facts in view that I propose to lay a scheme whereby, I think, it will be possible to determine, upon presentation of any case of urticaria, acute or chronic, what type of medication is likely to give the most immediate, complete and permanent relief. This can be accomplished by the simple expedient of determining the state of dermographism existing in the patient, which is easily done by stroking the skin with a smooth instrument.

Generally speaking, there are three types of dermographism commonly encountered: the white, or ischemic; the red, or hyperemic, and the elevated.

The white, or ischemic, dermographism appears to be a thyro-adrenal hypersecretory syndrome. The vasomotors are in a state of constriction. The basal metabolic rate is plus, and any drug that aggravates a hyperthyroid is contraindicated; therefore, such agents as adrenalin and ephedrin should not be used, for obvious reasons. In these cases I am in the habit of using pilocarpin and quinin, with most satisfactory results.

The red, or hyperemic, dermographism is a thyro-adrenal hyposecretory syndrome. The vasomotors are in a state of dilatation. The basal metabolic rate is minus and, of course, drugs which increase these secretions or substitute therefor, are the drugs of choice. In fact, they are imperative. I believe that adrenalin for immediate results, and thyroid extract as a fixing agent, so to speak, are productive of more good than anything that it has ever been my good fortune to use.

The third or elevated dermographism is also a thyro-adrenal syndrome, but while the basal metabolic rate of the ischemic is definitely high, and the hyperemic low, the elevated type, so to speak, is amphoteric. Some cases are hyper and some hypo. Owing to the fact that the elevated type appears to be more of a mass involvement, and the superficial vasomotors (if they exist), appear to be less involved than in the other types, it is not an easy matter to determine, offhand, the direction of the dysfunction, because the vasomotor response to stroking is not so marked as in the other types.

In these cases, therefore, it is necessary to obtain the basal metabolic reading for guidance. No one should treat a case of chronic urticaria without first having the basal rate determined.

I recently had two cases in point: one, an elderly man who had had an angio-neurotic edema, recurring at frequent intervals for the previous two years. His basal metabolic rate was minum 28 per cent. He was put on thyroid extract and his edema promptly vanished.

The other was a young man who had had an angio-neurotic edema for six months. For some reason his physician had put him on mixed treatment, and he became definitely worse. His basal metabolic rate was taken, and found to be plus 7 per cent. His pulse was fast and generally he presented evidences of hyperthyroidism. He was given pilocarpin and quinin, and his edema promptly vanished.

These cases have been under observation now for several months, and they still think they are well.

And here I wish especially to stress the matter of the time factor in the therapeutics of endocrin disturbances. That this is most uncertain and slow there is little doubt.

Endocrin therapy, generally, is directed towards the reestablishment of a normal physiological habit. I believe it is an accepted fact that habit formation with tissues and organs, as with individuals, is fairly easy to acquire but hard to control, and from its very nature endocrin therapy demands a period of treatment varying from a few weeks to possibly several years.

In closing I wish to say that while the measures I suggested have served me well, I am not at all convinced that there may not be other agents more potent than these.*

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*The literature on this subject is very scant. In April, 1902, I reported three cases of chronic urticaria as having been cured by thyroid extract. I did not, at that time, realize that different types meant anything. I treated them on the principle that "pigs is pigs" and "hives is hives" (New Orl. Med. & Surg. Jour., April, 1902).

Several have been reported since, but none prior this.

Nicholsky, in 1916, called attention to the lack of discrimination in the text books between red and white dermographism, but he offered no solution to the problem. (Abst. J.A.M.A., p. 179, 1916.)

D. M. Green, Los Angeles, Cal., in a paper entitled "Angio-neurotic Edema, a Pluriglandular Hypersecretory Syndrome," said that, being of thyroid and adrenal origin, angio-neurotic edema should be known as thyro-adrenal urticaria, but did not suggest that the dysfunction might be either hyper or hypo, as it really is. ("Essays on the Internal Secretions," Harrower, p. 273, 1920).

Metabolism observations in a case of urticaria scripta indicated an endocrin imbalance with thyroid chiefly involved. (Rowe and McCrudden, Boston Med. & Surg. Jour., July, 1924).

DISCUSSION (*Abstract*)

Dr. H. H. Hazen, Washington, D. C.—Yesterday we heard that urticaria is exclusively a matter of allergy. Today Dr. Roussel claims that the endocrin glands are responsible for practically all cases.

It is a question whether it is possible to classify all cases into one group. When we recall, for instance, that the presence of animal parasites in the body is frequently responsible for urticaria, it seems impossible to state that there can be but one cause. We should remember, too, that of late there has been much written in the *Journal of the American Medical Association* to indicate that appendicitis and gall bladder disease are responsible. In the allergic group there are numerous causes. Pollen, animal extracts, dust, household products, foods, bacterial substances, and clothing, may be exciting causes.

In all allergic states there is a predisposing cause. Mental or physical fatigue are probably the greatest causes, although diseased conditions, notably focal infection, may be responsible.

A number of years ago I made an attempt to learn the cause of sixty-one cases of chronic urticaria. Some of these may, or may not, have been due either to appendicitis or cholecystitis. Drugs are responsible in several instances and scabies for fifteen of the cases. It is not unusual to find urticaria following scabies, probably due to the absorption of protein from dead bodies of the parasites.

It was difficult to prove a hyper-susceptibility to food, but in three instances nuts were responsible. In one sad case the ultra-violet ray was used where there was a physical allergy to light.

Regarding the question of the relationship of urticaria to the ductless glands, I agree with those who believe that we know comparatively little regarding the functions of these structures and that all ills that the human is heir to should not be laid to these poor, overworked structures. It was good to hear Dr. Roussel insist that the basal metabolic rate should be determined in all instances; for this is a comparatively scientific procedure, even though the results are sometimes inaccurate.

All of us know that suprarenalin has a very marked, though frequently transitory, effect upon the lesions of chronic urticaria. Apparently the other drugs quoted by Dr. Roussel also have a comparatively good effect.

The point that I particularly wish to emphasize is that urticaria is probably of multiple causation and that in a high percentage of cases it is probably due to the absorption of protein from focal infection. Predisposing causes are also of importance.

Purely mental disturbances may be responsible for the outbreak. Those of us who knew Dr. Hartzell recall his story of the patient who had a violent outbreak of hives just as soon as he came into the presence of a new physician.

I should like to mention as a therapeutic aid in certain of the intractable cases the use of the ultra-violet lamp carried to the point of a severe erythema. However, in many communities, including Washington, D. C., it is extremely unsafe for a physician to use treatments of this kind. So long as we have shyster lawyers who are drumming up trade at the expense of the physician, and while juries are of the present type, it behooves every physician to be careful. In all probability, the next year or two will see many physicians employing only those methods of treatments which are absolutely safe, regardless of their curative effects upon the patient. Only those who have no property in their own names can afford to do otherwise.

Dr. Grafton Tyler Brown, Washington, D. C.—It is my opinion that some form of sensitization is the underlying factor in most cases of chronic urticaria. An exhaustive history is of prime importance, as by careful questioning the causative factors can frequently be elicited. It is not uncommon to see patients who, from their own experience, know that eating certain foods will produce urticaria, and yet cutaneous and intradermal tests with these same foods are completely negative. Someone has recently attempted to explain this discrepancy by stating that the reaction in urticaria takes place in the small blood vessels rather than in the cells of the skin. Positive skin reactions are relatively infrequent in urticaria, in comparison with asthma, hay fever, and eczema. In testing these patients, even slight reactions are worthy of consideration, and the substances giving such doubtful reactions should be retested at some future time. If they again react they should be considered etiological factors until proven otherwise.

Dr. Hazen called attention to the fact that foods are not the only substances that can cause chronic urticaria. Urticaria may be due to sensitization to any of the animal epidermals, pollens, drugs, or bacteria. I recall a patient with bad eczema and urticaria who gave positive reactions to the proteins of egg and silk. Elimination of egg in all forms from her diet, and the substitution of cotton and rayon clothing for silk, brought relief.

Urticaria may be due to the taking of drugs such as aspirin of quinin, and yet these drug-sensitive individuals rarely give positive skin reactions.

All foci of infection should be carefully sought, and when found treated surgically, or by autogenous vaccines, or a combination of the two. The stools should be examined for excessive putrefaction, or the presence of abnormal micro-organisms such as *Streptococcus hemolyticus*. Abnormal intestinal micro-organisms frequently originate in some focus of infection in the upper respiratory tract, and are swallowed. Acidophilus therapy, supplemented when indicated with autogenous vaccine, is probably the best treatment for abnormal intestinal flora.

Basal metabolism determinations are extremely helpful in selected cases. Hyperthyroidism is probably best treated by x-ray, and hypothyroidism, by the oral administration of dessicated thyroid in gradually increasing doses, until the metabolic rate has been brought to normal, which may be judged very largely by carefully watching the temperature and pulse.

Blood calcium determinations should be routinely done on patients with chronic urticaria, and those patients with a calcium deficiency should be given calcium and parathyroid by mouth and quartz mercury vapor lamp radiations.

Dr. L. W. Ketron, Baltimore, Md.—I would like to call your attention to some remarkable work which has been published by Sir Thomas Lewis and his associates, in a book called "The Blood Vessels of the Human Skin and Their Responses." It is particularly interesting to those who are studying urticaria and allied reactions in the skin. They have worked out an explanation for these reactions, which they believe

to be due to a histamine-like substance. The book is well worth reading by any dermatologist.

Dr. Jack W. Jones, Atlanta, Ga.—We are carrying the impression away of how little we really know about urticaria. The more we study it, apparently, the less we know.

For some time past we have gone over our urticaria patients from the standpoint of internal medicine. When we are all through then we have not found anything.

Sensitization tests in my own experience have been absolutely worthless. I have gotten nowhere with them.

I read a paper last year on sodium thiosulphate. After another year's experience it is still the best treatment that I have found for urticaria. It falls down at times, but it certainly has given me help.

Dr. J. R. Allison, Columbia, S. C.—I wish to call attention to two points: (1) there are very few cases of urticaria in which definite allergic causes can be determined and which can be cured by allergic methods of treatment, notwithstanding the claims of those who specialize in allergy. (2) I have found recently two cases of urticaria due definitely to nicotine in excessive smokers. I have not heard nicotine mentioned as a cause of urticaria.

Dr. E. S. Lain, Oklahoma City, Okla.—I, too, am convinced from my own experience that all urticarias are not due to any one single cause, though perhaps they are due to several causes. This has been frequently demonstrated. We are more or less inclined to call every chronic pruritic eruption which has papular lesions that we cannot definitely classify under any other name, urticaria.

I have within the past year seen two cases of early pernicious anemia which had previously been treated for chronic urticaria because of the generalized pruritis which is not uncommon in anemia. We should not lose sight of the fact that many other diseases may cause a generalized pruritis in certain stages which simulates urticaria.

Treatment is not under discussion, but I wish to ask Dr. Roussel if he can classify one of my cases in which arsphenamine gives immediate relief. A few years ago a general practitioner suggested to me to use arsphenamine in chronic urticaria. I gave my patient arsphenamine and three doses cured him of an urticaria of more than three years' standing. I find that Dr. Hazen had previously suggested this same treatment in certain chronic cases.

How may we classify cases which can be temporarily or permanently relieved by the administration of intravenous arsenic?

Dr. George M. MacKee, New York, N. Y.—Dr. Roussel's paper shows very clearly that urticaria, because of its polymorphous etiology, is not a problem limited to any one branch of medicine. However, the dermatologist is better acquainted with the varied clinical manifestations and the various etiological possibilities than is any other physician or specialist. Therefore the disease as a problem, both therapeutically and etiologically, really belongs to dermatology, and it should be investigated by dermatologists cooperating with immunologists, internists, bacteriologists, or other specialists.

Dr. Cosby Swanson, Atlanta, Ga.—Several years ago Dr. Roussel gave me his present classification of urticaria. The two classes were, as described today, those suffering from hyperthyroidism showing the hyperemic or red line, and those suffering from hypothyroidism showing the ischemic or white line.

At that time I decided to check up on a few chronic cases of urticaria by taking the metabolic reading. Of the ten patients in which the metabolic reading was made all confirmed his classification. When one is called upon to treat cases with chronic urticaria, this rapid method of ascertaining whether they are hyper or hypo thyroid cases is valuable. Those showing hyperthyroidism are very sensitive to the action of adrenalin, while those showing hypothyroidism require more than the average dose of the drug.

Dr. H. M. Robinson, Baltimore, Md.—Did I understand that Dr. Roussel doubts that certain factors may suddenly cause an allergic reaction after an individual has been in contact with them all his life without ill effect?

Quite frequently I come across patients who have received from eight to forty doses of arsphenamine or neo-arsphenamine, who do not react to the first or even the second injection, but after several doses "break out" with urticaria. These patients are then removed from the arsphenamine schedule for about two weeks, and after this period injections are again started with about one-third or less of the original amount, gradually increasing the dose, without causing a further outbreak of urticarial lesions.

We have had patients who have been coffee drinkers for many years, who quite suddenly developed a sensitization to this substance, with the appearance of eczematous lesions, and only on the removal of the coffee (when this has been diagnosed as the cause by history or by cutaneous test) does the patient become clear of the reaction. These patients' symptoms have disappeared so quickly upon the removal of the irritating substances that I have not attempted to determine the basal metabolism.

I should like to ask Dr. Roussel how quinin and pilocarpin act to cure urticaria.

Dr. Roussel (closing).—How are you ever going to cure a man of urticaria and know that he is well, if you remove from him the only thing that causes it?

I stated in my paper that anything in the world will cause urticaria, but I do not believe that it occurs in a perfectly normal individual. Where the endocrin "hook-up" is normal we have no urticaria.

We are talking about chronic urticaria, a pure intrinsic neurosis. We are not talking about dermatitis caused by ivy-poisoning or bug bites!

Serum sickness is not chronic urticaria, and is not generally relieved by the same methods. Intravenous 30 per cent sodium citrate (10 c. c. to adults) will stop this condition.

That the luetics are relieved by arsphenamine I am quite certain. Likewise, I think a bad tooth, infected tonsils, and gall bladders, and in fact all foci of infection, should receive proper attention.

MORE RECENT DEDUCTIONS IN OPEN BONE OPERATIONS*

By W. S. NASH, M.D.,
Knoxville, Tenn.

The topic of open bone operation for fractures has been a live one for many years. It does not yield to friendly discussion or strong verbal tilts any more than thyroid surgery, or surgery of the upper abdomen. The subject is yet wholly in the realm of doubt, notwithstanding the generous amount of discussion it has had recently. Since railway surgeons are specialists in the larger centers of population, and have frequent opportunities of performing open bone operations and comparing results with those obtained by other surgical methods, their experience in this line of endeavor should be commanding.

I cannot offer the solution of this surgical problem, but call your attention to a few outstanding facts in the consideration of it. These facts are grouped under two heads: those which favor, suggest or recommend to the surgeon the open bone operation; and those which discourage, deter or otherwise render it impracticable.

For many years I have been a student of end-results in bone surgery, but my analysis of cases has not always led me to conclude that the best results were obtained in a group of fractures. The conclusion is forced upon me that more might have been done for the unfortunate one and the maiming might have been lessened had the surgeon been bolder or a better mechanic. When you see poor end-results coming in from every quarter, your mental attitude is changed about the treatment of fracture, especially complicated fractures, or fractures partially diagnosed. Fixed methods and routine treatment give way to practical research and more radical treatment of fractures in the hope that better end-results may be obtained and that usefulness may not be impaired.

Complicated fractures are the residual of a fracture series after all the simple, comminuted or compound fractures have been completely diagnosed and classified. Complicated fractures are the ones that require the closest application of professional knowledge and the keenest sense of surgical judgment. The personal equation is very important in dealing with fractures, especially in determining their classification and the final operative interference. The thinking man, the man of knowledge, the careful man, the man of experience, sees fewer complicated fractures in a fracture series than does a poorly qualified, inapt, or inexperienced physician. Fractures must be closely studied and carefully diagnosed in order to be correctly classified and properly treated.

In dealing with all fractures, a definite diagnosis should be made, the position of fragments should be known, complications, if any, should be carefully studied, and the treatment should be so comprehensive as fully to cover the situation and to give reasonable indication of a good end-result. The thoughtful surgeon makes use of all his knowledge and of all the material things to correct or reduce a fracture, and to maintain its position and alignment. But in the exercise of his judgment and the application of his skill, without making use of the open bone operation, he many times fails; and failure in a fracture series, failure in union, fibrous union, poor co-aptation, no co-aptation, and poor alignment are always serious consequences.

The open bone operation is a surgical achievement of necessity, and rarely ever an operation of election, though there are a few outstanding cases which suggest an open bone operation from the very beginning. An analysis of more than one hundred fractures as they occurred in industry and transportation shows that 11 per cent are compound. These eleven cases should be considered as mandatory open bone operations, as they have all the indications involved in elected open bone operation, and more, towit: probable infection, severe lacerations of soft structures, comminutions, and hemorrhage. In consideration of these added dangers and the beautiful end-results often obtained in such open bone operations, should not we learn the lesson of more open bone work? Open bone surgery has been made a last resort by railway surgeons for some years for the following reasons: an extra hazard, probable infection, is added by attempting it, and criticism is encountered from members of the profession who are not skilled in the surgery of bones. In case of failure after the open bone operation, the consequences are greater and the likelihood of malpractice proceedings nearer. Aggression and experimentation are suggested by the open bone operation to the more conservative members of our profession.

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In an analysis of all that can be alleged against open bone operations, let me say that open bone surgery on fractured bones is not wayside surgery; it should be done by skilled and gifted men in the best regulated hospitals and under the strictest aseptic precautions. If this line is to succeed it will be in the hands of the more skillful operators. With these, infection will be so rare as to be negligible.

That criticism is encountered in open bone work there can be no doubt. There are good surgeons who can, and good ones who cannot, do open bone work. There are men who are so situated as to have all the necessary hospital apparatus; who have a trained hospital interne staff, have good judgment; and again there are men who have the knowledge, judgment and ability but lack the facilities to do open bone work. Without the facilities, poor judgment is shown in undertaking such work.

In cases of failure in the open bone operation I doubt that the consequences are more serious, though it has been generally admitted by the profession that they are. Few failures are recorded when such operations are performed by skilled surgeons in well-equipped hospitals, and in the event of failure there is this satisfying thought: that all has been done that could have been done, and the results, while not gratifying, are better than they would have been by any other method.

Inquire into the end-results, look into the x-ray laboratories, and you will find the accumulated evidence which justifies the conclusion that more open bone operations should be performed. Bad end-results, loss of function, maiming and deforming, are seen everywhere, in factories, in offices, upon the streets, and in your office. Call upon a skillful roentgenologist and in his laboratory and in his dusty files he will show you hundreds of skiagraphs of frightful end-results, due to lack of surgical knowledge, lack of surgical fortitude, lack of suitable facilities for treating and operating upon fractures. In these days of rapid transportation, the sum total of fractures is enormous, especially in automobiling, where fright, recklessness, and great momentum are encountered. Multiple fractures and compound fractures are very common.

I have analyzed the objections to the open bone operation. Let us set up the surgical achievement, the things that suggest, support and commend the more radical and more efficient treatment of fractures. The open bone operation should be undertaken when all other methods have failed: in irreducible fractures impinging upon delicate organs; fractures that sacrifice function; fractures that grossly impair usefulness; fractures that are frightful in appearance; fractures that are grossly deforming.

In fractures of the vault of the skull with unyielding compression; fracture of the zygomatic process of the temporal bone or supermaxillary bone (flat head or flat face deformity); fracture of the spinous processes of the vertebrae with compression symptoms; fracture of the patella or fracture of the olecranon; fracture of the head of the radius where usefulness alone is to be considered; close-end fractures and splitting fractures of the long bones; in none of these should you refuse an open bone operation.

I do not suggest that an immediate open operation should be done on all fractures. The cases themselves will suggest to you the proper open bone operation at the proper time. Many cases unpromising at first sight yield gratifying results by the open bone method in the hands of skilled surgeons. This plea is made that surgeons may turn their attention to, and make a more careful study of open bone operations; that poor end-results, maiming, unsightliness and deformities may be lessened and that the injured may ultimately be restored to perfect economical value to our people. If surgery stands for anything at all it stands for better work in the fracture realm. As it is now practised the results are disappointing.

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DISCUSSION (*Abstract*)

Dr. Edward T. Newell, Chattanooga, Tenn.—I do not think we should contemplate or attempt an open bone operation unless we have had wide experience in bone surgery. Cutting down on a bone is something that should not be undertaken lightly. One should have not only wide personal experience, but proper team work, adequate armamentarium, and hospital facilities. Unless you have the equipment and a trained team you are not going to get the results that should be obtained in bone work.

One of the indications for open operation is a limb or an extremity that has been so greatly traumatized that further manipulation in attempting reduction would produce more trauma to the soft part than to cut down on it.

If a child falls from a tree or is thrown out of an automobile and sustains a fracture of the elbow with the classical backward displacement, the proximal end of the humerus pressing forward on the brachial artery, producing swelling and ecchymosis, cutting off the

circulation to the forearm, it would be much easier and better to cut down on the fracture. Lift the distal fragment forward and over, suturing it in place with heavy chromic catgut. This is preferable to further traumatization of the elbow by manipulation in attempted approximation. Of course it is useless to say that you cannot put such an arm up in the classical Jones' position. A right angle or less is all that can be obtained at this time.

In fracture cases with great trauma, especial attention should be paid to the soft parts first. Later, the fracture can be approximated.

Dr. Fred H. Albee, New York, N. Y.—A man who is going to do operative fracture work must understand the physiology of muscle pull and the manipulations of the fracture table. If one has proper facilities and the knowledge, technic and dexterity to do the work, the question of infection will be almost nil. Many cases are damaged by manipulation.

We have to think of the blood vessels, nerves, tendons and muscles that may be injured from repeated manipulations. A good surgeon with a good fracture table may put those bones together with infinitely more delicacy by the open operation than by constant and repeated manipulations.

THE IMPORTANCE OF THE TIBIALS IN THE PRODUCTION AND RECURRENCE OF CLUB-FEET*

By J. WARREN WHITE, M.D.,
Greenville, S. C.

At the recently established Shriners' Hospital in Greenville, we are not satisfied with the results which are being obtained in the treatment of club-feet, and I am presenting this paper in the hope that by the exchange of ideas we shall be better able to obtain satisfactory correction.

Of course we have little difficulty with the relatively mild cases that respond easily to the simple plaster cast method when the children are still infants. It is the more extreme cases that are bothering us. The method that we are employing is the one in more or less general use and consists in the application of plaster casts at weekly or fortnightly intervals, the foot being gently manipulated between plasters before a new one is applied in a more corrected position. Instead of changing the casts weekly we have been "wedging" them on the alternate weeks, thereby, we believe, effecting a saving in time and material and making for better efficiency in the correction.

In the more difficult cases we have found that, in obtaining the necessary overcorrection, we are prone to produce two conditions which are the real cause of our dissatisfaction. First, in correcting the adduction of the forefoot and general varus, we are able to correct the varus of the os calcis before we have obtained what we desire in the forefoot, and by the time we have obtained what we call a satisfactory position of the forefoot we have a badly pronated tarsus. To avoid this condition, we sometimes stop before we have corrected completely the varus of the forefoot and our result is an unsightly "S"-shaped foot. Sometimes, when this condition is extreme, we have to resort to the so-called Ober operation, which has for its object the freeing of the tight deltoid ligament and other resistant ligamentous structures on the inner aspect of the foot in the tarsal region. This is a rather radical procedure and we hesitate to do it unless it is absolutely necessary. It has been our policy first to correct and pay especial attention to the varus and then to attack the equinus element. The second of our previously mentioned difficulties is a complication of a similiar nature, being the production of what we call a "rocker-bottom" foot. In other words, this is a convex surface of the lateral plantar region, the apex being at the distal end of the os calcis or the calcaneo-cuboid joint. In the bad cases we find that this frequently develops in spite of the careful lengthening of the tendo Achillis. There is something else besides this structure that is holding the anterior part of the os calcis down. X-rays of the ankle joint do not show the mortis between the internal and external malleoli to be too narrow to include the astragalus as Whitman* describes, which he believes is cared for by his malleotomy operation. The tremendous leverage obtainable on the front of the foot, in correcting the equinus, does correct the forefoot, but the os calcis and cuboid are held down so rigidly that the foot really breaks in the mid-tarsus. The "rocker-bottom" deformity is the result.

The above two conditions are the ones that have caused us to try to analyze the situation in an effort to find the etiology. Probably we shall be accused of using too much force and hurrying, but the same conditions have developed when we have been particularly slow and painstaking about the application and fitting of the plasters, trying to hold the heel where it

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*Whitman, R.: Orthopedic Surgery, 6th Ed., p. 766, Lea & Febiger, 1919.

belongs and preventing the valgus position. In asking the questions of ourselves what structure in the normal foot can cause it to assume the posture called club-foot, we cannot attribute it to any other element than the tibial muscles. Some actively contracting structure must be the cause, and it is difficult for me at least to believe that the ligamentous structures on the inner aspect of the tarsus can play any but a secondary part. I believe that an analogous condition can exist in the foot to a Dupuytrans contracture of the hand, for I have recently operated upon one; but it does not occur often enough to be considered an important factor in the every-day club-foot. It must be some active powerful agent and what else could it be but a muscle group? Of course after weight-bearing has been started, no one can deny that this is a factor in increasing the deformity in the severe cases.

One theory of the etiology of congenital club-foot is the maintenance of the deformity *in utero* by the pressure of the uterus on the lower extremity in the club-foot position of the crouched fetus over long periods of time while the foot is growing most rapidly. This may be possible, but I consider it a secondary factor, comparable to the weight-bearing factor after the child starts walking. The familial incidence may be explained by the fact that the exact structure or rather shape of the tarsal bones, particularly the astragalus, is transmitted in the germ plasm just as are the shape and relation of bones of the face. It represents a predisposing, not a causative factor, for it is not the rule that more than one case exists in a family.

One theory, which as far as I know is original, depends on the rather peculiar innervation of the posterior tibial muscle. The only muscles other than the posterior tibial that the internal popliteal innervates are the soleus-gastrocnemius, flexor longus digitorum, and intrinsic muscles of the sole of the foot, all muscles which incidentally could supplement the action of the posterior tibial as the causative agent of a club-foot. All the other muscles of the lower leg are innervated by the external popliteal or peroneal, which is, as we know, along with the ulnar at the elbow, the most exposed nerve of the body. I believe it is more than a coincidence that all the muscles that would act to produce a club-foot position, except the anterior tibial, are innervated by one well-protected nerve, and all the opposing muscles are innervated by another nerve which is peculiarly exposed to trauma even *in utero*. It would not be difficult to imagine a well developed fetus, temporarily in a breech position exposed to pressure of the maternal iliac crest at the proper point just below the knee at the point where the peroneal nerve passes around. Even in the vertex position the costal edge or the bodies of the vertebrae might cause a temporary pressure, enough to result in a transitory foot-drop paralysis, which in turn in the presence of predisposing conditions such as the shape of the astragalus, might be sufficient to start a club-foot deformity. This is highly speculative, but not entirely irrelevant.

In discussing the importance of the tibials in the production and recurrence of this deformity, let us consider, for a moment, the elements making up the condition. Talipes equino-varus describes it well except that there is another element present that is not included in this name. I refer to the internal rotation of the foot as a whole on the leg. In my opinion this means that there must exist some force attached to, but outside of the foot, and attached to the leg above where the rotation takes place, twisting the foot in. Here again I admit that, after weight-bearing is started on the deformed foot, this may be an added factor in causing a further rotation. Before weight-bearing, I see no possibility of there existing any other force than muscular which could effect this, and I can see no other muscle capable of doing it than the posterior tibial. Possibly the long flexors of the toes may be in a position somewhat to aggravate its action.

The part played by the anterior tibial in a club-foot, I believe, is secondary to the posterior muscle of the same name. However, I must admit that I have seen some bad club-foot deformities, the result of poliomyelitis, where the foot was practically flail with the exception of the anterior tibial. In these cases it will be noticed, however, that the equinus element is missing, as well as the internal rotation of the foot on the leg.

If one dissects the various insertions of the posterior tibial and sees for himself how tendinous prolongations go to every bone of the tarsus and all but the first and fifth metatarsus, occasionally even to the former, one cannot help being impressed by its efficient arrangement as an invertor of the foot and as an agent in producing a varus. Its attachment to the os calcis and cuboid after passing around the internal malleolus shows how it acts as an extensor of the

foot along with the soleus-gastrocnemius; and, as was stated previously, possibly explains why it is difficult to correct the equinus element even after generous lengthening of the tendo Achillis. If one recalls its origin from the upper half of the fibula and tibia and that it twists the foot inward in addition to inverting it, it is reasonable to believe that this action is responsible for the rotation element of the deformity, except where weight-bearing plays a part.

Therefore, it can be understood why I have been directing particular attention to the posterior tibial in the problem of club-foot. Inasmuch as it will take at least two years to come to any worth-while conclusion, and as I have been in Greenville little more than a year, and as we have been working on this particular problem only half of that time, this paper will take the form of a preliminary report.

I am not in a position to make a thorough search of the literature in Greenville, and so I have found it necessary to call on Dr. Albert Key, of St. Louis, in charge of the research department of the Shriners' Hospitals, for assistance. He reports that, after a thorough search of the literature, he has been able to find no reference to any discussion of the tibials in their relation to club-feet.

The procedure that is being followed at the Greenville Unit in certain typical cases of rather bad club-feet is to take only cases with symmetrical bilateral deformities. I have selected this type of case so that we may have reliable controls. Where there is a slight difference in the feet, special attention is directed toward the one considered the worse. In order to rule out as well as possible the personal equation, my associate, Dr. Clegg, carries on in the routine manner with the control, realizing that, when one is after a certain result one unconsciously expends more effort on the particular foot that is especially being worked on. The few cases that have been done, however, are encouraging and, in my estimation, indicate that this is a legitimate procedure. So far we have done about eight cases.

The technic is very simple. One goes in through a semilunar incision about the internal malleolus and comes down directly on the posterior tibial as it lies close to and immediately posterior to this bony landmark. It must be carefully distinguished from the tendon of the flexor longus digitorum which lies a little deeper and posterior to it. Ordinarily there is no difficulty in finding it if one keeps close to the malleolus. Distal to that point it is situated much deeper and its identification is not easy. At the malleolus, it could only be confused with the flexor longus digitorum, which can be identified by a slight pull and by watching the toes. After certain identification of the posterior tibial I remove a centimeter or so of the tendon right at the malleolus. I remove a section to prevent any reattachment. The wound is then closed and the usual club-foot plaster routine is adhered to. In very extreme conditions where the deformity is bad and the foot turned markedly in, the malleolus is deeply situated itself and the identification of the tendon is more difficult. I never encounter any real trouble if I stay near the malleolus. I never disturb the anterior tibial as it is too powerful a dorsiflexor to jeopardize.

In conclusion, then, it is only natural to suppose that the correction of severe cases of club-foot is facilitated by the elimination of an agent that is continually opposing the restoration, and which, in my opinion, is the real cause of the deformity. It has been said that once a club-foot is thoroughly corrected there is little chance for it to recur. I believe that, even after thorough anatomical correction of some club-feet, recurrence will take place because of the imbalance existing, namely: the lack of tone or power of the group of muscles innervated by the peroneal nerve and the greater relative strength of the posterior tibial. In such instances the cause of recurrence is as stated and cannot always be laid on the old alibi that the deformity could not have been fully corrected. The apprehension that I have is as to whether the permanent elimination of this muscle will predispose toward a really pronated foot later on. Let me emphasize the fact that this procedure is being carried out only upon difficult cases, young enough to have little bony deformity.

DISCUSSION (*Abstract*)

Dr. J. H. Kite, Decatur, Ga.—I cannot agree with Dr. White thoroughly on his idea of the origin of the club-foot, and question how much effect the posterior section of the tibial has in the treatment. I think of club-foot as a primary congenital defect. The child has inherited a tendency to it rather than a tendency for normality. We find in every four or five of our cases that some ancestor has had club-foot, also associated dislocated congenital hips, which it would be difficult to explain as a secondary development.

The difficulty we have in correcting club-foot is probably due to firm ligaments in the club-foot position. If you cut not only the tibial but all the

members, you still would have difficulty molding it back. I have never cut a posterior tibial, as the cutting of other tendons has been of little aid in helping me to correct club-foot.

We are treating cases of "rocker-bottom" deformity with plaster cast and wedgings, and have overcome some of the difficulties by wedging the club-foot in dorsal flexture. If we put the apex of the width at the heel, the foot would break. We put on the cast, molding it snugly to the heel, the point of motion being just below the heel line, then take the wedge out. When you flex the foot closely the cast sticks to the heel. You do not get the "rocker-bottom," and you can stretch out the para-knee.

The cases in which we have done without ether, using merely plaster cast and wedgings, have given flexible feet. The cases that have been anesthetized, in which we have broken up the cartilage on the feet, are more or less stiff and rigid.

Dr. Guy W. Leadbetter, Washington, D. C.—About a year ago, in Washington, we started to work out the etiology of club-feet on a purely neurological basis. We noticed in testing some of these patients that the peroneals showed a very sluggish reaction to the electric current, whereas the tibials and the posterior groups reacted rather sharply. We felt that that was a fairly good indication of some neurological change. We were fortunate enough to secure one or two still-born infants who had club-feet. We felt that there might be some delay in myelinization of the nerve trunks, and we made sections and dissected the nerves out in their entirety. We found no evidence of trauma over the head of the fibula.

We also made sections of the spinal cord. We have not been able to demonstrate that there was any change from the normal in the development. We have not yet reached the anterior horn cells with differentiating stains. We feel that possibly something there will give us a clue.

Ten cases all showed sluggish electrical reactions on the weak group of muscles. One of these still-borns had a very wide open spine.

I was interested in the traumatic theory of the disease, but do not see quite how it can explain the other deformities.

Dr. Wm. S. Baer, Baltimore, Md.—I have had a feeling for a long time that the majority of cases of club-foot have a neurological basis. On two or three occasions we have made sections of the lower part of the spinal cord. The cord may not ascend as it does in a normal child.

Until we know the normal embryological development of the extremities, we have nothing to compare the abnormal development with. There is a beautiful opportunity to study the pathological development of the limbs, which I believe will result in finding out the etiology of club-foot. We cannot leave out the development of the cord as the probable cause of club-foot.

Dr. A. Bruce Gill, Philadelphia, Pa.—I agree with Dr. Baer in his opinion as to the etiology of many cases of club-foot. Routine x-ray examination of a series of cases has shown that spina bifida occulta is frequently present. Of course, it is well known that many cases of frank spina bifida present this deformity of the feet.

In the treatment of club-feet in young children I have always been conservative and I do no operate. Effort is made to secure correction by manipulation with or without a general anesthetic as conditions of the feet demand and by the application of a series of plaster casts. It is seldom necessary to give anesthesia more than once or twice. At each application of a cast it is usually found that better correction can be secured than at the time of the application of the preceding cast. It is my opinion that this method of treatment results in more flexibility of the foot after correction than is obtained by operative measures.

Dr. White (closing).—I realize that the spinal cord and spina bifida could play an etiological role in club-foot. However, I wonder why it is that a club-foot deformity occurs frequently, and the other deformity rarely.

Dr. Leadbetter spoke of deformities other than club-foot. In my experience these conditions are not nearly so difficult to treat, or as prone to recur as the deformity under discussion.

I do not understand just what Dr. Kite meant by primary congenital deformity, unless it is the inherited predisposition I mentioned in the paper. In a certain portion of the cases I agree that this might be the start of a club-foot condition, but I believe that it occurs very rarely.

I have never thought of wedging the foot from behind, and while I cannot see how it could be so efficient as wedging in front, I shall try it.

Dr. Gill spoke of the administration of anesthetic in primary manipulation of the foot. In Greenville, unless we do an actual surgical procedure, such as a tendon lengthening, we have found that an anesthetic is not necessary.

A COMPARISON OF METHODS FOR THE DETECTION OF THE TUBERCLE BACILLUS*†

PRELIMINARY REPORT ON FIVE HUNDRED EXAMINATIONS

By Roy R. Kracke, B.S., M.D.,
Emory University, Ga.

In 1882 Robert Koch[1] established the etiology of tuberculosis and at the same time announced a reliable staining method for its identification and study. This, the acid-fast stain, has received many modifications since that time and was notably improved by Ziehl[2] and Neelsen, the Ziehl-Neelsen method, with various modifications, being employed to the present day by a large majority of laboratory workers.

There is probably no staining method in use today that gives such reliable and satisfactory results as the acid-fast stain, especially when positive results are obtained. When applied to

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†From the School of Medicine, Department of Bacteriology, Emory University.

specimens that contain a sufficient number of tubercle bacilli for demonstration it has no peer in efficiency.

In the routine examination of sputa for the detection of the tubercle bacillus there seems to be a field that is not covered by the acid-fast stain, this being the large number of specimens that contain such a small number of organisms that the chance of finding them is remote, and it is within this range that we find that type of case of pulmonary tuberculosis that gives every clinical evidence of the disease, yet in which the laboratory is unable to confirm the diagnosis by a positive finding in the sputum. For years the method of guinea pig inoculation has been employed, but this has proven unsatisfactory in many instances due to the number of organisms required to produce an infection in the animal, the time required for infection to set up, and the expense involved.[3]

In 1908 Uhlenhuth[4] announced his antiformin method for concentrating the specimen. He used a mixture of sodium hydroxide and sodium hypochlorite which liberated chlorin and destroyed many forms of bacteria, but left the tubercle bacillus unaffected. This method, or one of its modifications, is routinely employed in many hospitals with varying results. An acid-fast stain on the concentrate is the one commonly used, or the sediment may be planted on one of the various media that have been advocated for the growth of the tubercle bacillus.

In 1915 Petroff[5] announced a new method of detecting the tubercle bacillus which consisted essentially in concentrating the specimen with sodium hydroxid and planting the sediment on gentian violet egg medium. The principle involved was the destruction of the contaminating organisms, chiefly the gram-negative ones,[6] by the sodium hydroxid and the inhibitory action of gentian violet upon the gram-positive ones. This method has had considerable use since that time, but still does not result in a sufficiently high percentage of positives and low percentage of contaminations to be of outstanding value. Petroff had obtained 69 positive cultures from 69 specimens, and in another series[7] reported 129 positive cultures in 135 examinations. Corper,[8] however, found only 27 per cent positive in 526 examinations by this method.

In 1928 Corper[9] reported experimental studies in which he had inoculated tubercle bacilli intravenously into animals and made subsequent examinations of the various body tissues, using the acid-fast stain and guinea pig inoculation. He concluded that a minimum of 100,000 organisms per cubic centimeter of tissue is required before they can be detected by the acid-fast stained smear. He also made a similar observation regarding sputum, based upon the examination of sputum after the inoculation of the specimen with definite quantities of tubercle growth.

For years I have called the attention of my students to the lack of delicacy of the ordinary acid-fast stain on the untreated specimen. This can be shown in another way, on a mathematical basis. The average smear of tuberculous sputum on a three-by-one-inch slide averages about 2 x 3 cm., or 600 square mm. The area of the average microscopical field using a 10x ocular and 1.2 oil immersion objective with a 950 magnification is 0.02 square mm. The 600 square mm. area of the average specimen divided by the 0.02 mm. of the microscopical field indicates that there are about 30,000 microscopical fields in the smear.

The average laboratory worker when examining a slide for twenty minutes will cover about 500 fields. He therefore examines about one-sixtieth of the smear. One cubic centimeter of sputum will contain about 150 loopfuls of sufficient size to make a thin, even smear that would be desirable for examination. This was determined by weight. Therefore, one loopful of smeared sputum contains about 30,000 fields, and 1 c. c. about 450,000 fields. There would, then, have to be an average of 450,000 tubercle bacilli per c. c. to average one per field. So an individual could have 10,000 tubercle bacilli per c. c. of sputum and the laboratory worker could search through 500 fields and have the chance of finding the one organism that would be in that area.

An examination method that requires the presence of more than 10,000 organisms per c. c. of material examined could be justly considered inefficient.

So we should be constantly striving to find a more delicate method for the detection of the tubercle bacillus. When Corper announced a new method for this purpose in 1927[10] and again in 1928,[11] I decided to run a series of examinations to determine whether I could corroborate his work. The results are given in this paper in a preliminary form.

Corper's method consists essentially in using 6 per cent sulphuric acid as the concentrating agent, and planting the sediment on crystal violet potato medium to which has been added 1.5 c. c. of glycerine bouillon.

Lowenstein[12] was the first to suggest the use of an acid instead of an alkali as the concentrating agent, and stated that it was an efficient germicide for contaminating organisms and had little effect on the tubercle bacillus. Pawlowsky,[13] at the Pasteur Institute in 1888, first used glycerine potato, while Petroff[5] introduced the idea of using gentian violet in Dorsetts' egg medium. Corper[10] has shown that 6 per cent sulphuric acid is about the proper concentration, and he conceived the idea of employing crystal violet with the potato-glycerine bouillon medium.

In this series of examinations the method employed consisted of carrying out an acid-fast stain on the specimen, concentrating by the sulphuric acid method, doing an acid-fast stain on the concentrate and planting the sediment on crystal-violet-potato medium. The technic is as follows:

One c. c. of the specimen is ground in a sterile mortar with an equal quantity of 6 per cent sulphuric acid until the mass is homogeneous. It is then placed in a sterile centrifuge tube, incubated for 30 minutes at 37° C., washed with sterile saline solution, centrifuged, and the sediment planted on the potato medium. In carrying out this series I made an original acid-fast stain from the specimen selected and from the concentrate. Thus the results are those of three examinations, the primary smear, the concentrate smear, and the culture.

The results are incorporated in the following outline, based on three methods:

Number of specimens examined (sputum)	218
Number of contaminations	104
Number uncontaminated	114
Positive by direct smear	25%
Positive by concentration	12%
Positive by culture	27%
Positive by culture, negative by other methods	10%
Negative by culture, positive by other methods	14%
Positive by culture, negative by primary smear	12%
Negative by culture, positive by primary smear	11%
Positive by either of three methods	42%

Based on two methods (culture method omitted)

Number of specimens examined (sputum)	218
Total number of positives	37%
Positive by primary smear	30%
Positive by concentration	12%
	Specimens
Positive by primary, negative by concentrated	55
Negative by primary, positive by concentrated	14

From these results it is evident that the culture method possesses little advantage over the primary smear as carried out by this method. First, the problem of contamination is paramount. Almost 50 per cent of all cultures showed a growth of contaminating organisms, these being in almost all instances moulds. The growth of moulds usually appeared in from two to four days and in two instances moulds appeared after the growth of the tubercle bacillus. The cultures were sealed with a film of celloidin by the method suggested by Fried[14] and were certainly air-tight. There is little doubt that the contaminations were introduced at the time of seeding the tubes. It is interesting that all of these moulds were gram-negative, hence there was no inhibitory action by the gentian violet. Such a high rate of contamination is sufficient to prohibit the use of the sulphuric acid method of concentration.

In the 114 uncontaminated specimens the positive results in 27 per cent of the cultures, as contrasted with 25 per cent positive by the primary smear, offers such slight advantages that the method is not recommended for the average laboratory worker. My opinion is that the method of concentration is at fault, and to verify this I concentrated twenty specimens with the antiformin method and the same specimens by the sulphuric acid method. I had no contaminations in the former group and eight contaminations with the latter.

During the course of this work it sometimes happened that I was unable to work on the specimens until a few had become dried out. These I carried through in the same manner to observe the effect of drying. The results follow:

Number of dried specimens examined	35
Number of contaminations	33%
Number uncontaminated	66%
Number of growths of tubercle bacilli	30%
Positive by primary smear	29%

It seems that the drying of the specimen affects the viability of the organism none at all, as the percentage of growths obtained slightly exceeds that of the moist specimens. This is in accordance with current knowledge regarding the effect of dessication on the tubercle bacillus.[15] It is quite certain that drying of the specimen retards the development of contaminating moulds. As a whole, the drying process seems to be of benefit prior to this method of examination.

From the percentage of positive results obtained from the concentrated material, and the low percentage of positives on the cultures, I am inclined to believe that the fault lies in the method of concentration. The medium seems to be excellent for growth, as attested by the cultures on demonstration. From the work carried out so far I would recommend that the antiformin method be substituted and the crystal-violet potato medium be used. From the few cultures I have run it seems to be far more efficient than the acid method.

CONCLUSIONS

(1) Corper's method for the detection of the tubercle bacillus, using sulphuric acid concentration and crystal-violet potato medium, was unsatisfactory.

(2) The fault of the method apparently lies in the method of concentration.

(3) In a series of 218 sputa from clinically diagnosed pulmonary tuberculous patients, in only 42 per cent could the organism be demonstrated by either of the three methods.

(4) The crystal-violet potato medium is an excellent medium and suitable for growing the tubercle bacillus, because of its sensitivity, inhibitory effect on gram-positive organisms, and ability to give a rapid growth of the tubercle bacillus.

(5) It is suggested that either the sodium hydroxide or antiformin method be used for concentration and the above medium be used for growing the organism.

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DISCUSSION (*Abstract*)

Dr. George S. Graham, Birmingham, Ala.—It is highly desirable that we secure some practical method that will give us a certain identification of the tubercle bacillus in the shortest possible time. Dr. Kracke's figures demonstrate strikingly how readily we may miss a microscopical demonstration of the organism in cases showing definite clinical signs of pulmonary tuberculosis. There is a real need for a dependable cultural method that will involve not too much labor and that will give us an early and trustworthy determination.

Thus far we continue in our own laboratory to use Petroff's sodium hydroxide method for concentration and in doubtful cases, guinea pig inoculation for proof of the organisms. There are dangers, of course. I have seen guinea pig inoculations turn out badly because the sodium hydroxide was allowed to remain too long in contact with the tubercle bacillus. It is not permanently alkali-fat. But for routine work we shall not dare for the present to discard the animal inoculation method. The number of organisms that will affect the guinea pig is very small, while as Dr. Kracke demonstrates, the number needed for microscopic demonstration is large, and there are still difficulties to be surmounted in developing a satisfactory cultural method. Animal inoculation necessitates a delay that is sometimes most annoying, but if we can hand the clinician at the end of five or eight weeks definite results from guinea pig inoculation I think we are as near to giving positive results as it is today possible to give.

Dr. Alfred Blumberg, U. S. Veterans' Hospital No. 60, Oteen, N. C.—This is a subject which is of great interest to all laboratory workers, especially because once ever so often there is a hint of the accusation of superficiality from the physician when the laboratorian fails to find tubercle bacilli in a given specimen.

In our institution where the number of patients varies between 1165 and 600, we often have had difficulty in demonstrating tubercle bacilli in sputa. Several reasons may be responsible for this. A patient who previously has had a small hemorrhage is often afraid to cough, as he fears the recurrence of a hemorrhage. This patient's specimen, when submitted, may not represent a satisfactory sputum. Every morning I see from five to fifteen patients. Each of these comes to the laboratory, where he rinses his mouth and then expectorates into a sputum cup. I have noticed that many patients do not know how to expectorate. Many of them will furnish nasal-pharyngeal secretions, and such specimens of course will be unsatisfactory.

The obtaining of satisfactory growths frequently depends upon the individual worker rather than the culture medium employed. In a local institution one employe was unable to get more than six growths out of 100 sputa inoculated, whereas another employe using the same culture medium obtained growth in ninety instances from the same specimens.

As Dr. Graham stated, under certain conditions we cannot well get away from employing guinea pigs for the purpose of confirming the presence of tubercle bacilli. When results are unsatisfactory the trouble is frequently due to the early examination or autopsy of the guinea pig. A six weeks' period of incubation is not always sufficient for the development of definite evidence of tuberculosis within an animal. When a specimen contains comparatively few organisms, it will take from nine to twelve weeks before satisfactory evidence of tuberculosis is found. It is better, therefore, to wait from nine to twelve weeks before the animal is examined, rather than have to explain months afterwards, when the patient shows definite manifestation of tuberculous disease.

The finding of tubercle bacilli in the spinal fluid has been simplified since alcohol has been added to the spinal fluid. I have found it more practical first to add a drop of human or horse serum to the spinal fluid to be examined. After shaking the mixture I add alcohol

and centrifuge the material. Smears are made from the sediment and the smears, after proper fixation, are stained in the usual manner. The results are surprisingly good, as organisms present will be found at least within twenty minutes.

We have succeeded in working out a method in which the steaming process is omitted. The smears are exposed to an eight-minute fixation with 40 per cent formaldehyde, after which they are stained with carbol fuchsin without employing heat. The usual steps of decolorization and counter-staining follow. The results are most gratifying.

Dr. Charles Phillips, Richmond, Va.—I should like to confirm Dr. Blumberg's experience about the identification of tubercle bacilli in spinal fluid by the method of adding an albumin of some sort.

The alkali centrifugation has certainly, in our experience, given the best results.

Shaw brought out several years ago that if you will partially collapse or damage one lung of a guinea pig and then introduce subcutaneously or intraperitoneally the tubercle bacilli, the organisms will search out the damaged lung and produce the ordinary lesions very much sooner than if you inoculate them in the usual way. I have personally seen those lesions develop within ten days and kill the guinea pig. If we can thus diminish the time of waiting for the hungry, eager clinician, we shall certainly have gained an advantage.

FRACTURES OF THE SPINE*

By HUGH THOMPSON, M.D.,
Raleigh, N. C.

Your attention is invited to this subject because spinal injuries seem to be decidedly on the increase as a result of automobile and construction accidents; because such injuries present an important surgical, as well as medico-legal, problem; and further, because there appears to be wide divergence of opinion among surgeons as to the best methods of handling such cases.

Fractures of the spine readily fall into two main groups: (1) those accompanied by paralysis, and (2) those without paralysis.

In group 1 we find all manner of variations, from cases with complete severance of the cord to those with only a slight transitory paralysis from contusion or hemorrhage. Some of these patients will recover under conservative treatment, while others are hopeless from the start, regardless of what is done. Between these two extremes there are many cases that should be susceptible of considerable benefit from proper surgical treatment. If the cord is actually crushed or torn, no improvement can be expected because it is a well established fact that cord tissue has no power of repair. On the other hand, any amount of paralysis may be due to pressure from bone or blood clot, without actual destruction of the nervous tissue. According to Frazier, "The symptoms of a complete transverse lesion of the cord do not invariably indicate a transverse lesion."

Now, if it be a fact in any given case that the paralysis present is due to pressure and not to actual destruction of cord tissue, it seems reasonable to suppose that the more quickly and completely the pressure is removed, the better is the chance for recovery. It is true that if one waits a day or a week the clinical picture may change and so simplify the diagnosis. On the other hand, the golden opportunity of benefiting the patient may be lost by the delay. Pressure sufficient to produce paralysis may, if continued, produce destruction of nervous tissue. Scudder says:

"The prognosis without operation is grave. While the results of operation are not brilliant, they are sufficiently encouraging to warrant us in making the practice more general."

My own experience with cases of this sort has yielded no brilliant results, and so I am in need of improving my methods. I believe, however, that improvement should be sought by way of quicker and better operations, rather than by increased conservatism. Contrary opinions are expressed by some other surgeons who maintain that the results of laminectomy are not good enough to justify the risk, except in a few selected cases. My position is that the condition of these patients is such as to justify any reasonable risk, if the slightest hope of recovery is offered. In a personal communication, Dr. H. G. Turner, of Raleigh, reported a case in which complete paraplegia, including both sphincters, followed a fracture of the second and third lumbar vertebrae. He did a laminectomy within twelve hours of the accident, removed pieces of bone and blood clot causing pressure, and the patient made a complete recovery. This case looked like a transverse lesion of the cord on admission, and, had operation been delayed, such a result could hardly have been achieved. I believe this to be a very unusual result, because most cases of complete paralysis show an irreparable injury to the cord, but it proves the possibilities. In cases with only partial paralysis early operation should yield a considerable number of gratifying results.

Fractures of the spine in which no paralysis

*Read in Section on Bone and Joint Surgery, Southern Medical Association, Twenty-Second Annual Meeting, Asheville, North Carolina, November 12-15, 1928.

occurs represent a much larger and in many ways a much more important group of cases. Minor fractures; that is, those involving only spinous or transverse processes, present points of interest, but for the sake of brevity will not be considered here. Compression fractures of the bodies of the vertebrae, with or without involvement of the laminae and articular processes, are the important fractures of this group, as well as of group 1.

These fractures are usually produced by forced flexion of the vertebral column, one vertebra being caught and compressed between two others like a nut in a nut-cracker, and, like the nut, it may be only slightly cracked or may be completely crushed, depending on the force. Fractures of this type may occur in any part of the spine, but are more common near the dorso-lumbar junction. One of the remarkable features of these cases of compression fractures without paralysis is the fact that mild cases require practically as much treatment as severe ones. This is because any compression which is severe enough to produce fracture also crushes the blood vessels throughout the major portion of the body of the vertebra in question. Therefore, bony repair is slow and cannot even begin until new blood vessels have been developed. During the early weeks the vertebra is therefore relatively soft and readily compressed further.

"Compression fracture of the spine may develop delayed symptoms. Post-traumatic spondylitis (Kummell's disease), developing as a result of compression fracture of the spine, is a distinct entity." (Scudder).

Prevent Kummell's disease by recognizing and adequately treating compression fractures of the bodies of the vertebrae (Frederick Jones, *J.A.M.A., December 1, 1923.*) This being true, the importance of not overlooking mild compressions is at once apparent. The lateral x-ray, I believe, invariably shows the condition, but antero-posterior views cannot be relied on. Quite a few cases have come to my attention in which no lateral views were taken and in which the fracture was completely overlooked. In some of these untreated cases I have been able to secure the antero-posterior views which were taken shortly after the accident, and compare them with antero-posterior views taken two months or more later. Definite increase in the vertebral collapse was evident, this having been brought about by unprotected weight bearing before consolidation had had time to take place.

In view of the foregoing, there seems no room for doubt on one point; that is, that the injured vertebra should be protected for a long period of time following the accident. The form of protection and the length of time seem to be open questions. The writer feels that there is no satisfactory substitute for recumbency and insists on this for from seventy-five to ninety days. A plaster shell may be used or simply a hard bed. The main thing is to keep the patient flat. After this rest period the patient is allowed up in a spinal corset or brace, which is worn for from nine to eighteen months.

In cases which show any appreciable deformity this is first corrected as much as may be by traction and manipulation, the patient being prone and traction applied to head and feet. A plaster shell is always used in such cases.

Following this line of treatment, our results have been good. Cases treated include both men and women; laborers, housewives, trainmen, a trained nurse, and others. All have been able to resume their former occupations and in the main there has been little complaint from residual pain or weakness.

In view of results which are satisfactory, one hesitates to change the plan of treatment. Yet it is highly objectionable from the economic standpoint, and any improvements that could be made would be in order. A review of the literature shows every sort of variation. Schiesl[1] recommends getting these patients up in plaster jackets after ten days or so in bed, and reports good results. Mulleder[2] recommends doing some sort of spine fusion operation almost routinely soon after the accident, and thereby claims to cut the period of disability in half, and to get better results than are seen under conservative treatment. An analysis of the case reports on which Mulleder's conclusions are based seems to indicate that his patients undergoing spine fusions received very much better aftercare than those treated conservatively.

It is my belief that spine fusion operations will shorten the period of disability, but they are too serious to be undertaken without very good reasons, and if good results can be secured by conservative treatment, a few months more of wearing a spinal brace is a very much simpler and safer procedure than a spine fusion. In cases where symptoms persist or where observation indicates that the fractured vertebra is collapsing, such an operation is clearly indicated.

CONCLUSIONS

(1) Fractures of the spine, complicated by extensive paralysis, whether complete or not,

should have exploratory laminectomy at the earliest possible moment which the patient's general condition permits.

(2) Compression fractures of the bodies of the vertebrae without paralysis should be treated by correction of the deformity, followed by prolonged recumbency, followed by a spinal brace for one year or more.

(3) Spine fusion operations should not be done routinely in compression fractures, but should be reserved for those cases which do not recover under conservative treatment.

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DISCUSSION (*Abstract*)

Dr. Wm. A. Boyd, Columbia, S. C.—Before the introduction of the Queckenstedt test it was my practice to operate immediately upon all fractures of the spine associated with paralysis. In that way only could it be determined whether or not there was actual destruction of the cord, or whether the paralysis was due to some remediable cause, and we had no way of determining when the period of compression passed into one of degeneration of the cord.

Since the introduction of the Queckenstedt test I operate upon only those cases showing a definite blocking of the spinal canal.

The shock of the operation I think is of very little moment. I have not seen any degree of shock from the operation that gave me any worry. In the last few cases that I have operated upon I used local infiltration anesthesia and did not find any appreciable increase in difficulty or time of doing the operative work.

There is a definite relationship between the ultimate result that you may expect in cases of fractures of the spine with paralysis, and the time from injury that they are operated upon, because if there is no actual division or destruction of the cord, and the paralysis is due entirely to pressure, then sooner or later, if that pressure continues, there must occur degeneration of the cord tissue.

In compression fractures of the spine conservative surgery is indicated. Only in unusual cases will operative surgery be needed, because even if you subject these fractures to a fusion operation or bone graft, they still must be kept recumbent for a definite length of time for fusion to take place, and if the individual is kept the same length of time recumbent and protected, he will develop his own fusion or bone graft. Recumbency, rest and protection are the essentials of treatment in these cases and not operative surgery.

There is an increasing number of accidents. In years back they were almost entirely among men, but now a large proportion of compression fractures occur in women because of the increased use of motor cars. The general practitioner and general surgeon should know that where an individual has received a severe injury to his back, certainly one associated with flexion of the spine, after which he complains of pain in the back, because a roentgenologist says there is no fracture, strips of adhesive plaster should not be applied and then the patient dismissed. The individual should be referred to an orthopedist or to some man capable of taking care of and protecting him for the future, because disability comes in the future.

Dr. Charles E. Dowman, Atlanta, Ga.—For many years I was at a loss to know when a laminectomy was indicated. One cannot rely absolutely on the neurological evidence of a complete transverse lesion. We used to have to rely on this, and felt that in those cases with a complete transverse lesion no operation was indicated. Operation was confined to those cases in which there was some retention of sensation below the level of the lesion. Since the introduction of the Queckenstedt test we have something that will give us a very definite indication as to whether or not a laminectomy is indicated. There is no object in doing a laminectomy unless there is something to remove. This something may be indriven bone fragments, blood clots, or edema.

We follow the advice of Coleman, of Richmond, and as routine do a lumbar puncture below the level of the lesion, attach a glass tube and read off the pressure in millimeters, and then do a bilateral jugular compression. If there is a prompt rise of the fluid in the tube and a prompt fall to the original level on release of the jugular compression, we have no block whatsoever. If there is a partial block, there is a step-like rise on jugular compression, and on release of compression there is a step-like fall to a level which is higher than the original level. If there is a complete block there is no effect whatsoever when the jugulars are compressed.

In doing the test one must be sure that the patient does not strain or cough, as that will produce a dilatation of the spinal veins, which in turn will cause a certain amount of rise of the column of fluid in the tube. If we have neurological evidence, therefore, of cord injury, and we have a total block, we do a laminectomy as a matter of routine. In doing a laminectomy, the dura should always be opened and left open.

If we have no evidence of spinal block we repeat the test daily for three to five readings, until we are satisfied that there has occurred no compression of the spinal cord.

There is just one other point that I would like to mention, because it has given us a great deal of help. This relates to the care of the bladder. Repeated catheterization, as we all know, usually leads to infection. The method as practised during the war, namely, that of allowing the bladder to overflow, is not satisfactory, as usually there is residual urine which leads to infection. As a matter of routine, in all cases of paralyzed bladder a suprapubic drainage by means of a Pezzar catheter is done. Should the bladder regain its function the catheter can be removed and the suprapubic wound will promptly heal.

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AFTER MIAMI, CUBA

"The loveliest land that human eyes have ever seen!"

"This land, O Most Serene Majesties, is so wonderfully beautiful that it surpasses all others in charm, even as the light of the day surpasses that of the night. * * * All my men declare that there could be no land more beautiful. Now, therefore, I am silent, for I realize that all I might say would be too little; let its merits be sung by those who have greater skill in language. I assure Your Majesties that, in my belief, there can not be better lands under the sun."

The first is said to be the exclamation of Christopher Columbus when on October 28, 1492, he discovered the Island of Cuba. The second is an excerpt from the official report of Columbus to Ferdinand and Isabel, King and Queen of Spain. To the Nineteenth Century world traveler these statements may seem extravagant. No doubt they are extravagant, but though they may be, there is infinitely more of truth than fable in these statements, for Cuba is a "lovely land," a land of beauty and charm.

Nearly four hundred years later (1871), a noted American writer, Julia Woodruff, in her book, "My Winter in Cuba," said:

"Go to Cuba—go, if you possibly can. In Cuba the language, the architecture, the landscape, the flora, the customs, everything is new, strange, suggestive. For us the life there becomes one continuous poem. You will live as in a country of enchantment, unable to tell which part of the vision is earthly and which belongs to the domain of the imagination * * *."

Those who have been privileged to visit Cuba, the Cuba of today, can with all sincerity give enthusiastic approval to the above statement, though it was written sixty years ago.

Following the Miami meeting of the Southern Medical Association in November, there will be the chance of a life time to see this "lovely land" of Cuba, under ideal conditions, and at a cost within the reach of all. A special rate has been granted from Miami to Cuba and return to Miami of $27.50 plus Pullman fare, leaving Miami Friday night, following the closing session of the Association, via the Overseas Railway, arriving Key West early next morning and then leaving on a palatial steamer of the Peninsular and Occidental Steamship Line for a never-to-be-forgotten voyage, which puts one in Cuba past mid-afternoon.

The first view of Cuba is of the mountains of Matanzas Province, then Morro Castle and gradually Havana, and as the boat proceeds the Presidential Palace and other beautiful buildings, the Prado, the Maine Monument and many other interesting places come into view. Entering the harbor, there is the spot where the Maine was blown up and soon thereafter the boat docks.

Havana, modern in many ways, with modern conveniences for the traveler, still maintains its original setting. The charm of old Havana lingers. A trip to Cuba is not complete unless one sees something of the interior. Splendid highways lead to many parts of the island. If one's time is limited so that only one interior trip is permitted, the trip to Matanzas, one hundred and twenty miles on a modern highway, is undoubtedly the trip of choice. On this trip may be seen the native life of Cuba in its purest form, one of the large sugar refineries, the beautiful Yumuri Valley, Bellamar Caves and interesting old Matanzas City. This is a drive the memory of which will linger long.

In Havana is the beautiful Casino, "the Monte Carlo of Cuba," where those who wish may "chase Lady Luck" to their hearts' content, leaving happier or sadder, perhaps the majority sadder. And Havana offers many brass rails for those who may wish again to put a foot on one. While a hundred dollars worth of purchases may be brought back duty-free, the liquid refreshments must be left behind.

Space does not permit a description of the many things of interest that await the traveler to Cuba. Havana and the interior of Cuba are worth seeing. They are worth four or five extra days and the price it takes to see them. One can take a four or five day trip to Cuba, be entertained at a good hotel and be amazed at the amount he can see in that length of time, at a cost of less than a hundred dollars, from Miami and back to Miami. The Association is negotiating with several touring agencies to conduct all-expense tours. The Southern Medical Association's meeting comes before the busy resort season, which will enable its guests to secure accommodations at the best hotels of Cuba at moderate rates, such hotels as the Sevilla-Biltmore, Plaza, Royal Palm, Ritz, Inglaterra, Regina, Perla de Cuba, Lincoln, Florida, and many others.

AMENORRHEA IN YOUNG WOMEN

An ovarian hormone which can be standardized has been available now for several years. The extract of Allen and Doisy[1] of the University of Missouri has the measurable effect of inducing heat or estrus in a spayed mouse, and the quantity necessary to produce this effect in a mouse of a given size is pharmacologically determined, and is called a mouse unit.

This substance ranks already among the few gland extracts of known physiological effect on animals. It has rather pronounced physiological and psychic effects upon women, though the nature and extent of these is little understood. Its induction of estrus in animals has been largely studied, but the relation of menstruation of human beings to estrus in animals is still unknown. There is no very strong *a priori* reason to believe that a drug which will induce estrus in a mouse will induce menstruation in a woman.

A number of women students at the University of Minnesota were examined and their basal metabolism was found to be 15 per cent below the standard normal.[2] Whether this is in any way related to the climatic and soil conditions which class Minnesota among the goiter states is not known. A number of cases of amenorrhea were observed, and all cases fell in the group with low basal metabolism. It was determined to treat some of them by injections of the ovarian hormone of Allen and Doisy. A nineteen-year-old woman who had not menstruated for five months was given a number of injections of the extract. In three weeks she menstruated for six days. Another woman of twenty-two who had had amenorrhea for five years received two series of injections. Five days after the last, her menses appeared and lasted two days.

These are merely further proofs of the specific activity of this drug. That it is advisable so to induce menstruation in young women is highly questionable. Perhaps thyroid medication, perhaps merely improvement of the general condition by rest, diet, and other measures, would have been better treatment. However, physicians using a new drug always confer a favor upon the medical profession by accurate observation and reporting of its effects.

1. Allen, Edgar; and Doisy, E. A.: An Ovarian Hormone. J.A.M.A., 81:819, 1923.

2. McClendon, J. F.; Burr, George; and Wildebush, F. F.: Induction of Menstruation in Women by the Use of Ovarian Hormone. Proc. Soc. Exper. Biol. and Med., 26:450, March, 1929.

FILTERABLE FORMS OF TUBERCULOSIS

Imagination is a scientist's luxury. Without it he is a plodder, a valuable contributor to the scientific literature often, but as was said of the ancient Saxons, a "dull brother of the ox." If he indulges in more than an occasional flight of fancy, and fails to check and recheck all flights, he ceases to be a scientist.

Certain bacteriologists in recent years have gone to an extreme, perhaps, in postulating many forms and cycles for the simple bacteria of disease. For years following Pasteur's discovery of the disease-producing organisms, it was taught that the ordinary bacteria, *Bacillus typhosus, Staphylococcus aureus, Spirocheta pallida, Bacillus tuberculosis,* were fixed in their morphological forms; that is, were always rods, cocci, or spirochetes respectively, and reproduced by fission only. Lately there has been a tendency to report cyclic variations for some of these, comparable with the cycles of malaria plasmodia and larger fungi.[1] The hypothesis has been advanced with much accompanying evidence that the well known laboratory bacteria may appear in different forms, perhaps a rod

1. Hadley, Philip: Microbic Dissociation. Jour. Inf. Dis., 40:1-312, 1926.

as a coccus, a visible microscopic form as an ultra-microscopic filter-passing one. It has pleased the imagination of many scientists, and a wealth of literature has tended to demonstrate these variations of species formerly considered fixed. Workers on the other side of the question have disputed each claim to the finding of morphological variations. Filterable, ultra-microscopic forms of the tubercle bacillus have been a number of times reported. The question of the existence of a filterable form has an important bearing upon the question of tuberculous infection of the fetus *in utero*.

The more distinguished the name which endorses any disputed doctrine, of course, the more likely is it to be accepted by a non-bacteriological audience. France has produced in the present generation many distinguished bacteriologists. Calmette, of the Pasteur Institute, has been particularly well known in this Country for his production of a vaccine for prevention of tuberculosis.[2] B.C.G., or *Bacillus Calmette-Guerin*, is a living bovine form of tubercle bacillus attenuated by growth on a bile medium. Calmette and his coworkers have administered it by mouth to many infants exposed to tuberculosis in the home, and excellent results in the prevention of tuberculosis by this means have been claimed. The work is mentioned merely to recall Calmette's standing as a bacteriologist of international reputation.

Calmette has also made a number of contributions to the literature of the filterable forms of the tubercle bacillus, which he calls the tuberculous ultravirus. A culture of living tubercle bacilli is filtered through a porcelain filter, which allows passage of none of the ordinary bacteria. Such filtration has frequently been used to sterilize liquids which could not be sterilized by heat. This "sterile" filtrate inoculated into a guinea pig produces, it is said, an atypical form of tuberculosis, from the lesions of which acid-fast rods are obtained only after prolonged searching. After passage through several animals this ultravirus was said to recover its virulence and normal form, and again to produce typical acid-fast tubercle bacilli and typical tuberculous lesions in the guinea pig.[3] Some degree of success in the vaccination of animals with this filterable form of the organism has been reported.[4]

These findings, although accepted abroad, have been questioned in America by several workers, who have made it their function to challenge many reports of filterable forms and mutations of ordinary microscopic organisms.[5] Frobisher[6] has filtered sterile broth with all possible aseptic precautions, and obtained bacterial forms similar to those reported as filterable. He concludes that filterable forms described by many writers are merely contaminations. Regarding the reported guinea pig tuberculosis from filtered cultures, the stand is taken that the organisms found in the guinea pigs were either tubercle bacilli which had accidentally passed through an imperfect or leaky filter, or they were timothy grass bacilli. Cooper and Petroff[7] report the finding of acid-fast bacilli, possibly timothy grass bacilli, in the glands of one-third of a number of healthy guinea pigs examined. They deny that the existence has been proven of a filterable form of tubercle bacillus.

It is fortunate for the physician that many bacteriologists are willing to devote time to the clarification of this subject, since the technic of bacteriological research is extremely difficult. The measurement of a distant sun or of the electrons of an atom of copper may be more readily and indisputably accomplished.

One is grateful for the establishment of the present laboratory methods for the diagnosis of tuberculosis, uncertain and capable of further improvement though they frequently appear to be.[8] They are the result of extended experimental efforts, of checking and rechecking and repetition of work by many searchers after truth.

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2. Vaccination of the Newborn against Tuberculosis with *Bacillus Calmette-Guerin*. Ed. J.A.M.A., 89:115, 1927.

3. Calmette, A.; and Valtis, J.: Filterable Elements of the Tuberculosis Virus. J.A.M.A., 92:2086, June 22, 1929.

4. Valtis, J.: et Saenz, A.: Les filtrats de voiles jeunes de cultures tuberculeuses (ultra-virus) exercent-ils un effet protecteur vis-a-vis de l' infection tuberculeure virulente? Comptes Rendu de la Soc. de Biol. 101:353, Juin, 1929.

5. Brown, J. H.; and Frobisher, M.: Technical Difficulties Encountered in an Attempt to Produce Bacterial Mutations. Bull. Johns Hopkins Hosp., 40:318, 1927.

6. Frobisher, Martin: On the Action of Bacteriophage in Producing Filterable Forms and Mutations of Bacteria. Jour. Inf. Dis., 43:46, 1928.

7. Cooper, F. B.; and Petroff, S. A.: "Filterable Forms" of the Tubercle Bacillus. Jour. Inf. Dis., 43:200, 1928.

8. Kracke, Roy R.: A Comparison of Methods for the Detection of the Tubercle Bacillus. The JOURNAL, this issue, p. 678.

Miami---Where We Meet

MOTOR HIGHWAYS*

Just as alluring as its golf courses, its gleaming, sandy beaches, its golden groves, are the velvet-smooth roads that radiate in all directions from Miami. Short motor tours, because of these broad, hard surfaced traffic arteries, are an integral part of the well-rounded vacation in Miami.

Miami's appeal is, naturally, directly to the motorist from the North whose home country embodies none of the scenic features of this tropical paradise. Quickly accessible to Miami are the scores of southern Florida cities and towns, through primitive jungles rich with the perfume of tropical flowers and even out across the watery reaches of the Atlantic, where on both sides of the road is the booming surf of bay and ocean.

By virtue of these roads Miami is tied in with all the remote corners of America's last frontier. Across the Everglades one finds travel on the Tamiani Trail, a panorama of beauty in its wild state. The trail itself brings civilization into the last haven of Florida birds and animals and the stolid Seminole Indian, whose home in the cypress hammocks calls to mind the swift tempo of that civilization. He travels yet in his carved canoe along the crystal clear streams of the Everglades, while not far off the white man, in cushioned ease and luxury, strikes the contrast he cannot understand.

Here the scenes change with the passing miles: a cypress swamp, a plateau, marsh land with a thousand birds flapping their wings in unison, a bald eagle lifts itself gracefully off the roadside, and in the clear stream paralleling the roadside silvery fish leap and play in the sunlight.

Farther on westward one sees vast fields of truck produce, as he nears the west coast; sweet corn, potatoes, tomatoes, as far as the eye can travel.

But southward from Miami engineers have performed another feat in building America's most striking road, the Overseas Highway. It is over this stretch of approximately 175 miles that the motorist finds himself more in the role of navigator than autoist as he speeds over the only roadbed in the ocean. On either side is an emerald sea, while straight ahead the road stretches in endless miles to Key West, southernmost United States city.

On the Overseas Highway along the Atlantic Ocean.

The road joins the ragged keys together as a necklace of jewels. Some are populated sparsely, others are but a blot of tropic growth, but all afford striking scenery to the Northern motorist.

The Keys are noted, too, for their fruit, and it is from the lower Keys that the finest lemons and limes are shipped. There are also dozens

*By Charles C. Canny, Publicity Department, City of Miami.

of species of birds, more tropical than those of the Everglades and more like the flamboyantly-colored birds of Central America. From a point just south of Miami, almost to Key West, there is an area that is noted for its game fish and the inland salt water streams that ebb and flow between the Atlantic and gulf are excellent for boating and fishing.

Shorter tours, however, are in abundance in and around Miami, offering a variety of interesting points rarely found in an area equal to that covered by Miami and its contiguous suburbs. Fortunately for Florida, road building is a comparatively simple operation in the southern portion of the State, where the natural road material is the coral rock on which the city is built.

Across the bay or along the Atlantic Ocean, through beautiful residential districts and agricultural country, are roads that at once make motoring an uninterrupted pleasure. Beautiful estates, landscaped in tropical flowers and fruits, royal and coconut palm vistas, are always on hand to delight the newcomer.

The original type of architecture, developed in Miami from the Moorish, Mediterranean and Spanish, is striking in contrast to the construction of commonplace American cities. It is apparent from any of the dozen or more major highways that circulate through and about Miami proper.

Book Reviews

The Normal and Pathological Physiology of Bone, Its Problems. By R. Leriche, Professor de Clinique, Chirurgicale a la Faculte de Strasbourg, and A. Policard, Professor d'Histologie a la Faculte de Lyon. Authorized English Translation by Sherwood Moore, M.D., Professor of Radiology, Washington University School of Medicine, and J. Albert Key, M.D., Assistant Professor of Clinical Orthopedic Surgery, Washington University School of Medicine. 236 pages, illustrated. St. Louis: The C. V. Mosby Co., 1928. Cloth, $5.00.

Although small in size, this volume by Policard and Leriche represents the most comprehensive work on bone physiology at present available. Every conceivable problem in the biology and pathology of bone is given consideration. In many cases it has been possible only to state the problem, to review whatever work has already been done, and to point the way to the solution by suggesting the course of further research. Other problems have been brought closer to solution by original work of the authors.

Particularly admirable is their work on the histogenesis of bone and cartilage. They show that bone may be laid down in connective tissue which is in any stage of development, embryonic or highly specialized. The influence of this stage of embryonic development to the type of resulting bone is beautifully demonstrated. The adaptation of bone, the role of the periosteum, the resorption of bone; all are most interestingly treated. But this is not exclusively a laboratory book. The practical application of the principles of bone physiology is at once evident to the most mechanically minded surgeon. The authors have in their last chapter, under the title of "Future of Bone Surgery," suggested some applications of these fundamentals, the possibility of influencing callus formation, and of modeling new bone to adapt it to its function.

A better understanding of the failures and successes of bone grafting operations and of many other details of bone surgery will follow a study of the physiological principles here presented.

A complete bibliography of two hundred nineteen references, international in scope, adds to the value of the work.

Clinical Electrocardiograms. Their Interpretation and Significance. By Frederick A. Willius, M.D., Section on Cardiology, The Mayo Clinic, Rochester, Minnesota, and Associate Professor of Medicine, The Mayo Foundation, University of Minnesota. Quarto of 219 pages, with 368 illustrations. Philadelphia and London: W. B. Saunders Company, 1929. Cloth, $8.00.

This book is devoted to the purely clinical aspect of the science of electrography. The references to the underlying physical principles of the apparatus and the more physiological aspects of the subject have been omitted. Numerous curves are shown. The discussion is brief and practical. There is an excellent bibliography.

Very little mention is made of the effects of various therapeutic agents such as digitalis and quinidine on the electrocardiogram. This book should be of considerable value to the internist who is interested in the more elementary aspect of the subject.

Diseases of the Heart. By Henry A. Christian, M.D., Sc.D., LL.D., Hersey Professor of the Theory and Practice of Physic, Harvard University, and Physician-in-Chief, Peter Bent Brigham Hospital, Boston, Mass. Volume III, Oxford Monographs on Diagnosis and Treatment, Edited by Henry A. Christian. 355 pages. New York: Oxford University Press. Price per set of ten volumes, $100.00.

A distinguished clinician has summarized for the practitioner his views of an increasingly important group of maladies. The author has made important contributions to clinical cardiology. He has successfully combated the English dictum that the use of digitalis should be restricted to patients with auricular fibrillation.

The introductory chapter in which general statements regarding the problems involved are mentioned should be read by every medical student and every physician who treats patients with heart disease.

The most practical portions of the book, that is, those sections which deal with diagnosis and treatment, and which comprise most of the volume, are particularly good. The few pages which are devoted to the pathological physiology represent an expression of opinion and do not seem to be up to the standard of the remainder of the volume. The book is very readable and should be of particular interest to the general practitioner.

International Clinics. A Quarterly of Illustrated Clinical Lectures and Especially Prepared Original Articles on Treatment, Medicine, Surgery, Neurology, Pediatrics, Obstetrics, Gynecology, Orthopedics, Pathology, Dermatology, Ophthalmology, Otology, Rhinology, Laryngology, Hygiene, and other topics of interest to students and practitioners. By leading members of the Medical Profession throughout the world. Edited by Henry W. Cattell, A.M., M.D., Philadelphia, U. S. A., with the collaboration of Chas. H. Mayo, M.D., Rochester, Minnesota, and others. Volume I, 39th Series, March, 1929. Philadelphia and London: J. B. Lippincott Company.

All the excellent articles in this bound volume of current medical literature cannot be reviewed, but among those which deserve special mention are the following:

Dr. Lewellys F. Barker's clinics on hemiparesis, headaches and alcoholism are distinctly Barkerian, and therefore interesting and instructive. His scientific discussion of the clinical aspects of alcoholism is classical, but many physicians will not agree with his views regarding prohibition and the Volstead Act. Professor F. d'Herelle, of Yale, who was awarded a medal by the Philadelphia Pathological Society for his work on "The Bacteriophage," presents some startling results from his investigations in this new field of bacteriology. He is convinced that the behavior of the bacteriophage, which he terms "a parasite of the bacterium," decides the issue of life or death with patients who have cholera or other serious infectious diseases. Simon Flexner, in a brief article, summarizes the work of the Rockefeller Institute on the specific prevention and treatment of poliomyelitis. Stewart Roberts, of Atlanta, always interesting, discusses pellagra. He accepts the Goldberger theory of food deficiency and advises brewer's yeast in its treatment. No authority is better qualified to discuss amebiasis than Charles F. Craig of the U. S. Army, and his article on latent amebic infection is worth while. Held and Goldbloom, in an excellent treatise on pernicious anemia, suggest that since the introduction of the liver treatment the disease has lost its pernicious character, its name should be changed to Addison-Biermer's anemia. Vascular surgery is ably discussed by Ashhurst, and the concluding article by Col. Coupal, White House physician during Coolidge's administration, presents an excellent review of the progress of medicine during the year 1928.

A Manual of Diseases of the Nose, Throat and Ear. By E. B. Gleason, M.D., LL.D., Professor of Otology, Graduate School of the University of Pennsylvania. Sixth Edition, Thoroughly Revised. 12 mo. of 617 pages with 262 illustrations. Philadelphia and London: W. B. Saunders Co., 1929. Cloth, $4.50 net.

This is the largest little book known: light in weight, with an excellent index; wonderfully condensed and well illustrated. It has no superior as a text book for students and general practitioners. The specialist of extensive experience can find many valuable suggestions in the thirty-five pages of formulas. One is amazed that a book of this size can so thoroughly cover the subjects treated. The illustrations of instruments and details for the performance of the many operations are especially useful. The few references to authorities and the absence of footnotes rather add to the satisfaction one gets from reading a text of this elementary nature. The author claims it was written "to supply" students and general practitioners with the essential facts, and he has accomplished his purpose.

Surface Anatomy. By Arthur Robinson, M.D., F.R.C.S., Professor of Anatomy in the University of Edinburgh, and E. B. Jamieson, M.D., Lecturer on Anatomy in the University of Edinburgh. 173 pages, illustrated. New York: William Wood & Co., 1929.

This book is intended by its authors "to be used by the student while he is dissecting—and also while he is reviewing anatomy in the later stages of his course." It is an excellent reference work for use when it is necessary accurately to locate anatomical structures from more easily visible or palpable surface landmarks. For this explicit purpose the book holds a unique place in medical literature. It is the most complete and accurate work on the subject. As a text it is hardly practical because of the wealth of minute detail included.

Surgical Pathology. By William Boyd, M.D., M.R.C.P., Ed., Dipl. Psych., F.R.S. Can., Professor of Pathology, University of Manitoba; Pathologist to the Winnipeg General Hospital, Canada. Second Edition, Revised and Reset. Octavo of 933 pages, with 474 illustrations and 15 colored plates. Philadelphia and London: W. B. Saunders Co., March, 1929. Cloth, $11.00 net.

Boyd's second edition includes many changes and additions, but the general character of the book is unchanged. There are twelve chapters on general pathology and thirty-eight on special organs or systems. Material has been utilized which has direct bearing upon surgical tissue and surgical diagnosis. The author has exercised as much good judgment in what he has omitted as in what he has included. For example, the surgical pathology of the skin and eye have been excluded, since they are highly technical, would be voluminous and are structures not usually operated upon routinely by the general surgeon.

Splendid monographs have appeared on individual subjects in surgical pathology, but Boyd's book covers the ground more completely, accurately and satisfactorily than can be found elsewhere. Its compact size and condensed but specific data, together with many appropriate cuts, make the volume a boon to the surgeon and a joy to the clinical pathologist.

Syphilis. By Charles C. Dennie, B.S.M.D., Assistant Professor of Dermatology and Syphilology, University of Kansas School of Medicine; Chief of Heredosyphilitic Clinic, Childrens' Mercy Hospital; Chief of Syphilitic Clinic, Kansas City General Hospital; Dermatologist and Syphilologist, St. Luke's, Research, and St. Mary's Hospitals. 304 pages, illustrated. New York and London: Harper and Brothers, 1928. Cloth, $2.50.

The book is designed primarily to give the practitioner a knowledge of the fundamental facts of this important disease. The portions of the book dealing with the gross and microscopic pathology of the lesions in the various organs are particularly good. The clinical manifestations of the early stages of the disease are well described. One is inclined to wonder that the author has not devoted more space to the description of cardio-vascular syphilis, particularly as regards the early recognition of aortitis, a condition which is usually not diagnosed until grave complications have developed. Central nervous system syphilis also occupies a rather small portion of the book. In general, however, the volume may be said to contain practical information in regard to diagnosis and treatment.

Diseases of the Liver, Gall-Bladder, and Bile Ducts. By Sir Humphry Rolleston, Bart., K.C.B., M.D., Hon. D.Sc., D.C.L., LL.D., Regius Professor of Physic in the University of Cambridge; and John William McNee, D.S.O., M.D., D.Sc., F.R.C.P., Associate Physician to University College Hospital. Third Edition. 884 pages, illustrated. New York: The Macmillan Company, 1929.

For more than twenty years Rolleston's book has been a standard English text. During the fifteen years which have elapsed since the last edition, medical knowledge has been very much enriched by the studies of liver function. To this subject, as well as to the pathology of hepatic disease, the younger author has made important contributions. The combination of the two writers has been a happy one and has resulted in a modern work which retains the best features of the older editions. Despite the emphasis laid upon function, the pathological point of view predominates throughout the book.

The volume is encyclopedic in scope, is well indexed, and contains an exhaustive bibliography. It should serve as an excellent reference book for student and practitioner. The internist also will find it of great value, for it contains much that is new to him.

The Genesis of Epidemics and the Natural History of Disease. By Clifford Allchin Gill, Lieutenant-Colonel, Indian Medical Service, Director of Public Health, Punjab, M.R.C.S. (Eng.), L.R.C.P. (Lond.), D.P.H. (Eng.), D.T.M.&H. (Eng.), Fellow of the Royal Society of Medicine; Fellow of the Royal Society of Tropical Medicine; Chief Plague Medical Officers, Punjab (1909-1911); Chief Malaria Medical Officer, Punjab (1913-1923). 550 pages, illustrated. William Wood & Co., 1928. Cloth, $6.50.

Dr. Gill has devoted many years to the study of the natural phenomena which are associated with the spread of epidemics. His main thesis is concerned with the fact that epidemics occur only at a time when certain predisposing factors co-exist. The most important of these are:

(1) Diminished resistance in the population due to a period of comparative freedom from infection;

(2) Enhanced virulence of the infective agent; and

(3) Meteoric conditions which favor the rapid transmission of the virus.

He proves his thesis in a thoroughly convincing manner for malaria and cites much evidence to show that a similar hypothesis explains epidemics of bubonic plague and of influenza. The book should be of particular interest to health officers and epidemiologists.

Tuberculous Intoxications. By Joseph Hollos, M.D., Pathologist of St. Vincent's Hospital, Staten Island, New York. 132 pages. New York: William Wood & Co., 1928. Cloth, $3.25.

The two outstanding impressions gained from reading this book are, first: the author's belief in the message he is bringing to the profession; and next, that he is an extremist. He divides tuberculosis into cases which manifest symptoms and "tuberculous intoxications." He counts the latter the cause of a large part of human ills, including "rheumatism, epilepsy, peptic ulcer, and all forms of menstrual disturbances." He treats the patients with Spengler's immune blood and at times tuberculin. In his conclusions, his enthusiasm carries him beyond the results that others have had.

Edema and Its Treatment. By Herman Elwyn, M.D., Assistant Visiting Physician, Gouverneur Hospital, New York. 177 pages, illustrated. New York: The Macmillan Company, 1929. Cloth, $2.50.

Elwyn does not believe that the various types of edema can be explained adequately by physical chemical concepts. He points to many facts which seem to him to indicate that in all types of edema there is a disturbance of a water regulating center in the central nervous system. His evidence for this assumption seems to be inadequate, as it is quite possible that further advances in biochemistry and biophysics will clarify many of the details which the author now regards as obscure. He quotes a number of writers in support of his assumption that the volume of the circulating blood remains relatively constant in edematous subjects. Such an assumption as regards patients with heart disease is contrary to the every-day experience at the post-mortem table. Recent work from Germany and from Belgium supports the belief that in certain patients with edema the blood volume may be markedly increased, and constancy of this body function is an essential premise for the author's views of edema as a regulatory disturbance. It appears that his stand is untenable.

The more practical portions of the book, that is, those devoted to treatment, seem to be of higher standard than the rest of the volume.

The Tonsils and Adenoids and Their Diseases, Including the Part they Play in Systemic Diseases. By Irwin Moore, M. B., C. M. (Edin.), Late Honorary Surgeon to the London Throat Hospital for Diseases of the Throat, Nose, and Ear, Great Portland Street, and also to the Hospital for Diseases of the Throat, Golden Square, London, W. 395 pages, illustrated. St. Louis: The C. V. Mosby Co., 1928. Cloth, $6.50.

It seems strange that books on tonsils and adenoids and their diseases continue to appear. Even the laity has followed the specialist in excessive anxiety about tonsils. The author has devoted nearly four hundred pages to the subject and has accomplished his purpose exceedingly well. If one would criticize, it would be to say that he has given too many details and not sufficiently stressed his own preferred points of operative procedure. The book is well illustrated, has a splendid index, and the advice is sound and conservative.

Tropical Diseases. By Philip H. Manson-Bahr, D.S.O., M.D., B.Ch., D.T.M.&H., F.R.C.P., Lecturer at The London School of Tropical Medicine and Hygiene. Ninth Edition. 941 pages. 35 plates. 401 illustrations. 34 charts. New York: William Wood & Co. Cloth, $11.00.

Since the appearance of the first edition, this book has been accepted as a standard. Each section has been thoroughly revised and in parts rewritten. Throughout the book there is a note of conservatism and directions as to treatment are given with clarity. The book shows a great familiarity with advances made in tropical medicine, and the editor's accepted position in tropical medicine makes his own remarks authoritative.

The book has long been the "Osler's Practice" of the physician in the tropics and the present edition deserves the same confidence that was given it during Manson's life.

What Is Life? By Augusta Gaskell. Introduction by Karl T. Compton, Professor of Physics, Princeton University, and Raymond Pearl, Professor of Biology, The Johns Hopkins University. 324 pages. Springfield, Illinois: Charles C. Thomas, Publisher, 1928.

This is one of the most fascinating books the writer has ever read with the idea of offering a review. Assuming the correctness of the Bohr-Sommerfeld planetary theory of the atom, the author has produced a most alluring argument for her contention that life has resulted from a condition of a "critical concentration of ions" affording an opportunity for a stray electron to sneak into the field of positive hydrogen nucleus while its own electron is wandering off at "aphelion."

The book does not lend itself readily to a popular review, but it is of extreme interest to a reader with limited knowledge of physics but interested in the subject and eager to increase his knowledge. At first the idea seems a little bizarre and one almost unconsciously compares it with a family triangle where the flapper stenographer sneaks into the home and upsets the equilibrium.

The author has very wisely chosen a well known professor of physics and a teacher in biology to present introductions to the book, and while they do not endorse the theory presented, they have to admit that the "fundamental assumption must be admitted as possible." The reviewer has carefully read this volume, even to the third time, and always with increasing interest. To do the book justice would require many pages. No one, even with a meager knowledge of physics, can lay it aside once it is begun. In his "Philosophy" Bertram Russell says: "But now electrons and protons themselves are dissolved into systems of radiations by Heizenberg and into systems of waves by Schrodinger * * * *. And these are not wild metaphysical speculations; they are sober, mathematical calculations accepted by the great majority of experts." If this be true, the fundamental assumption on which the book is written is destroyed. But the reviewer will reread the book many times and, as Osler said, he preferred to be wrong with Plato and accept immortality, the reviewer prefers to be wrong with Millikan and accept the planetary theory of the atom, earnestly hoping that some laboratory workers may follow the suggestions of Dr. Gaskell and seek for proof or disproof of this most engaging hypothesis as to the fundamental cause of life.

Physiotherapy in General Practice and for the Use of Masseuses. By E. Bellis Clayton, M.D., B.CH. (Cantab.), Director of the Physiotherapeutic Department, and in Charge of the Massage and Electrical School, King's College Hospital, London. Second Edition. 231 pages. New York: William Wood & Co., 1928. Cloth, $3.50.

This manual presents physiotherapy in a broad sense. Not only are the methods of application of light and electricity but exercises are given in detail. Only a page and a half are devoted to massage, and there is no section on hydrotherapy.

Despite these omissions, the book is to be commended for its clarity of expression and the detail with which the indications and applications of physiotherapy are outlined in disease.

Notes on Chronic Otorrhoea, With Especial Reference to the Use of Zinc Ionization in the Treatment of Selected Cases. By A. R. Friel, M.A., M.D., (Univ. Dub.), F.R.C.S.I., An Assistant Aurist, School Medical Service, London County Council; Aurist to Tottenham, Hornsey, and Walthamstow Education Committees. 87 pages, illustrated. New York: William Wood & Co., 1929. Cloth, $2.25.

This is a small volume of ninety pages, with fair illustrations, describing and commending the use of electric current and zinc ions in the treatment of chronic otorrhea. One reads it with interest and remains unconvinced.

What Every One Should Know About the Eyes. By F. Park Lewis, M.D. 70 pages. New York: Funk & Wagnalls Co., 1928. Cloth, 30c.

For many years Dr. Lewis has been recognized as the leader in the educational campaign for the prevention of blindness. This little volume is short, less than seventy pages, can be carried in the vest pocket, and it would be a blessing if every high school and college student would read the first half of it once a year. In the campaign for a physical examination on every birthday one could wisely advise the occasional reading of a volume from the National Health Series, no one of which is superior to this by Dr. Lewis.

Diagnostic Methods and Interpretations in Internal Medicine. By Samuel A. Loewenberg, M.D., F.A.C.P., Assistant Professor of Clinical Medicine, Jefferson Medical College; Assistant Physician to the Jefferson Hospital. 1032 pages, with 547 illustrations, some in colors. Philadelphia: F. A. Davis Company, 1929. Cloth, $10.00 net.

This book is written from a practical viewpoint, the author explaining methods of elicitation and interpretation of physical signs in the diagnosis of disease. In special sections, laboratory methods are given. A highly commendable feature of the book is the way the author explains the indications for special examinations.

Southern Medical News

ALABAMA

Dr. W. E. Wilson, Dadeville, County Health Officer of Tallapoosa County, has been awarded a scholarship to Johns Hopkins University by the Rockefeller Foundation.

The Northwest Section of the Alabama Medical Association held a conference in Tuscaloosa on June 6 with the Tuscaloosa Medical Association. More than one hundred members attended.

The Northern Division of the Alabama Medical Association held its meeting in Gadsden on May 23.

Dr. W. Groce Harrison, Birmingham, has been appointed lecturer on the history of medicine at Vanderbilt University School of Medicine, Nashville, Tenn., and will assume his duties in September.

Deaths

Dr. W. H. Bell, Birmingham, aged 65, died May 21, after being ill several months.

Dr. Alvenzi Jasper Gilbert, Birmingham, aged 73, died March 21.

Dr. Charles Isaac Dahlberg, Suggsville, aged 69, died March 18 of heart disease.

Dr. Eugene Robinett Smith, Section, aged 85, died March 28 of cerebral hemorrhage.

ARKANSAS

The Polk County Medical Society has elected the following officers for 1929: Dr. F. A. Lee, Vandervoort, President; Dr. F. Q. McElroy, Mena, Vice-President; Dr. F. C. Mullins, Wickes, Secretary; Dr. B. H. Hawkins, Mena, Delegate to State Meeting; Dr. J. G. Hilton, Mena, Alternate.

Dr. C. W. Garrison, State Health Officer for the past twelve years, has been reelected for a four-year term.

Dr. O. L. Williamson, Marianna, and Dr. F. O. Mahanoy, El Dorado, have been reappointed as members of the State Board of Health.

The Randolph County Medical Society elected the following officers for the ensuing year: Dr. J. K. Loftis, Pocahontas, President; Dr. W. E. Hamil, Pocahontas, Vice-President; Dr. W. E. Hughes, Pocahontas, Secretary-Treasurer.

Dr. Paul Z. Browne, Hot Springs, formerly with the Martin Clinic, has removed his offices to the Dugan-Stuart Building.

The Arkansas Tuberculosis Association held its twenty-first annual meeting in Little Rock May 6-7.

Dr. W. A. Montgomery, Atkins, was seriously injured in an auto accident while en route to the State Medical Meeting at Hot Springs. Dr. G. C. Webb and Dr. W. P. Scarlett, of Russellville, were in the car with Dr. Montgomery, but they escaped with minor injuries.

The Arkansas Medical Society, at their annual meeting in May, elected the following officers for this year: Dr. Thad Cothern, Jonesboro, President; Dr. E. E. Barlow, Dermott, President-Elect; Dr. George B. Fletcher, Hot Springs, First Vice-President; Dr. B. H. Hawkins, Mena, Second Vice-President; Dr. J. G. Gladden, Western Grove, Third Vice-President; Dr. R. J. Calcote, Little Rock, Treasurer (reelected), and Dr. Wm. R. Bathurst, Little Rock, Secretary (reelected). Dr. Dewell Gann, Sr., Benton, was reelected Chairman of the Council, and Dr. S. J. Wolfermann, Fort Smith, was named Secretary of the Council. Fort Smith was selected as the 1930 meeting place.

Dr. F. Walter Carruthers, Little Rock, announces the opening of an office with the Medical and Surgical Clinic of Texarkana. He will be in Texarkana every two weeks on Saturdays only.

St. Bernard's Hospital, Morrilton, has elected Dr. Robert Caldwell and Dr. F. Walter Carruthers, both of Little Rock, as members of the hospital staff.

The Jackson County Medical Society has elected the following officers for this year: Dr. M. L. Harris, Newport, President; Dr. J. R. Loftin, Grubbs, Vice-President; Dr. K. K. Kimberlin, Tuckerman, Secretary; Dr. I. H. Erwin, Newport, Delegate to State Meeting, and Dr. J. B. Ivy, Tuckerman, Alternate.

Deaths

Dr. Victor H. Hallman, Hot Springs, aged 68, died March 11.

Dr. Silas W. Moreland, Jonesboro, aged 78, died April 29 of heart disease.

Dr. William C. Vandiver, Mena, aged 69, died May 12.

DISTRICT OF COLUMBIA

Dr. John A. Foote, Washington, has been elected President of the Medical Society of the District of Columbia.

Dr. Joel T. Boone, Washington, physician to President Hoover, was given a testimonial dinner recently by the Board of Trustees and Faculty of Hahnemann Medical College and Hospital, Philadelphia, of which Dr. Boone is a graduate.

Dr. Hubert Work, Washington, a Colonel in the Medical Reserve Corps, attended the International Conference on Military Medicine in Paris in May.

Dr. Arthur P. Noyes, formerly Assistant Superintendent, St. Elizabeth's Hospital, Washington, has been appointed Superintendent of the State Hospital for Mental Diseases, Howard, R. I.

FLORIDA

At their tenth annual meeting held in St. Augustine in April, the Florida Railway Surgeons Association elected the following officers: Dr. Harold D. Van Schaick, Jacksonville, President; Dr. W. E. Burnett, St. Augustine, Vice-President, and Dr. E. W. Warren, Palatka, Secretary-Treasurer. The meeting next year will be held in Pensacola on the day preceding that of the Florida Medical Association.

The Florida Heart Association was organized at the recent meeting of the Florida Medical Association and the following officers were selected: Dr. M. J. Flipse, Miami, President, and Dr. Louie Limbaugh, Jacksonville, Secretary-Treasurer. This Association will be affiliated with the Florida Public Health Association and the American Heart Association. Mr. Sherwood H. Smith, who is Executive Secretary of the Florida Public Health Association, was appointed Executive Secretary of this new association.

Dr. Oliver P. Broadbent, Jacksonville, announces the removal of his offices to 209-10 Wade Building, 1022 Park Street.

The annual meeting of the Florida East Coast Medical Association was held at Daytona Beach, June 14-15.

Dr. G. A. Longbrake, Fort Myers, has been appointed President of the Gorgas Health Corps of Fort Myers.

Dr. John Elwood Maines, Gainesville, and Miss June Gill, Lynchburg, Va., were married on May 4.

The annual report of the Department of Health, Miami, gives the birth rate per thousand of population as 14.63, the death rate 7.69, and the infant mortality rate 65.7. Miami has an estimated population of 140,000.

Deaths

Dr. Ralph Edward Kleckner, Miami, aged 43, died February 25 of uremia.

Dr. John McGready Oglesby, Bartow, aged 73, died April 6.

Dr. Edwin Lightner Nesbit, Deland, aged 50, died April 16 as the result of an automobile accident.

Dr. Wm. Marshall Richardson, Raiford, died May 1.

GEORGIA

The Medical Association of Georgia, at their eightieth annual meeting held in Macon May 10, elected the following officers: Dr. William R. Dancy, Savannah, President; Dr. George Y. Moore, Cuthbert, President-Elect; Dr. Charles H. Richardson, Jr., Macon, and Dr. Grady N. Coker, Canton, Vice-Presidents; Dr. Allen H. Bunce, Atlanta, Delegate to the American Medical Association, and Dr. Olin H. Weaver, Macon, Alternate. The next annual meeting will be in Augusta May 14-16, 1930.

Dr. Samuel H. Haddock, Macon, has been elected Health Officer of Jenkins County.

The Seventh District Medical Society has elected the following officers for 1929: Dr. C. V. Wood, Cedartown, President; Dr. J. S. Alsobrook, Rossville, Vice-President; Dr. W. H. Perkinson, Marietta, Secretary-Treasurer.

Dr. E. P. Quillian, formerly of Valdosta, has removed to Brunswick.

Dr. D. J. Buchanan, Savannah, has been appointed a member of the staff of the Veterans' Bureau, Cleveland, Ohio.

The Hall County Medical Society has named the following officers for 1929: Dr. C. J. Wellborn, Gainesville, President; Dr. H. S. Titshaw, Gainesville, Vice-President; Dr. Pratt Cheek, Gainesville, Secretary-Treasurer.

The Burke County Medical Society has elected the following officers for 1929: Dr. W. H. Sutton, Midville, President; Dr. J. B. Lewis, Waynesboro, Vice-President; Dr. W. Harry Sutton, Midville, Secretary-Treasurer.

The Warren County Medical Society has elected the following officers for this year: Dr. F. L. Ware, Warrenton, President; Dr. F. B. Ricketson, Warrenton, Vice-President; Dr. Robert C. McGahee, Warrenton, Secretary-Treasurer.

Dr. John D. Wiley, formerly of Milledgeville, has removed to Sparta.

Dr. H. H. Lancaster, formerly of Waycross, has removed to Hoschton, where he is associated with Drs. L. C. and M. B. Allen in the practice of medicine and surgery.

Dr. Hugo Robinson, Albany, has been reelected Commissioner of Health for Dougherty County.

Dr. Paul L. Holliday, Athens, announces the opening of his offices in the Southern Mutual Building, and will limit his practice to gynecology and obstetrics.

The Floyd County Medical Society has elected the following officers for 1929: Dr. A. H. Dellinger, Rome, President; Dr. J. H. Mull, Rome, Vice-President; Dr. C. H. McArthur, Rome, Secretary-Treasurer.

The Dooly County Medical Society has elected the following officers for this year: Dr. L. H. Bishop, Unadilla, President; Dr. H. A. Mobley, Vienna, Vice-President; Dr. F. E. Williams, Vienna, Secretary-Treasurer.

The Troup County Medical Society has elected the following officers for the year: Dr. H. McCulloh, Sr., West Point, President; Dr. W. R. McCall, LaGrange, Vice-President; Dr. H. McCulloh, Jr., West Point, Secretary-Treasurer.

The Baldwin County Medical Society has elected the following officers for 1929: Dr. U. S. Bowen, Milledgeville, President; Dr. W. F. Dobyns, Milledgeville, Vice-President; Dr. J. W. Mobley, Jr., Milledgeville, Secretary-Treasurer.

Deaths

Dr. R. L. Cheney, Shellman, died recently following a long illness.

Dr. John Boyd Rudolph, Gainesville, aged 56, died April 10 of heart disease.

Dr. Thomas Lorenzo Holcombe, Jefferson, aged 50, died April 4 of heart disease.

Dr. Frank Cole Baker, Fort Benning, aged 51, died April 12 of occlusion of the coronary artery.

Dr. Henry Parson Hamrick, Eatonton, aged 45, died April 23 of angina pectoris.

Dr. H. B. Lee, Luxomni, aged 42, died March 11.

Dr. Henry Grady Carter, Atlanta, aged 37, died April 4.

Dr. F. D. Nichols, Atlanta, aged 65, died March 4 of pneumonia.

Dr. William H. Rusk, LaGrange, aged 56, died April 10.

Dr. Alfred Christie, Brunswick, aged 65, died April 19 of cerebral hemorrhage.

Dr. Olin Estes Shankle, Commerce, aged 49, died March 31 of angina pectoris.

Dr. Oscar N. Pendergrass, Monroe, aged 57, died April 28 of pernicious anemia.

Dr. Henry Adams Smith, Americus, aged 27, died April 17 of Bright's disease.

Dr. Robin Adair, Atlanta, aged 50, died April 26 of injuries received in an automobile accident.

KENTUCKY

Dr. Charles K. Beck, Louisville, announces that he is now associated with Dr. J. H. Hester, with offices at 849 Starks Building, limiting his practice to eye, ear, nose, and throat.

Dr. Samuel M. Baxter, Louisville, is now associated with Dr. Octavus Dulaney, 811-18 Brown Building, practice limited to ophthalmology and otolaryngology.

Dr. Matthew David Garred, Ashland, and Miss Lottie Mae Rogers, Lexington, were married April 6.

Dr. John A. Sloan, Albany, and Mrs. Flossie Duvall, Wago, were married April 2.

Deaths

Dr. Thomas Rice Welch, Nicholasville, aged 69, died April 24 of burns received when his home caught fire.

(Continued on page 34)

FRACTURE COURSE for GRADUATES

The Fracture Service of the

MASSACHUSETTS GENERAL HOSPITAL

offers an

INTENSIVE SIX DAY COURSE

October 7 to 12, 1929, Inclusive

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THE course will extend through a period of eight hours each day divided into morning, afternoon and evening sessions. The instruction will be based on acute and convalescent fracture cases and on the study of known end results in over 800 old cases. It will include operative and non-operative treatment, demonstrations of splinting, suspension, and skeletal traction, and anatomical review.

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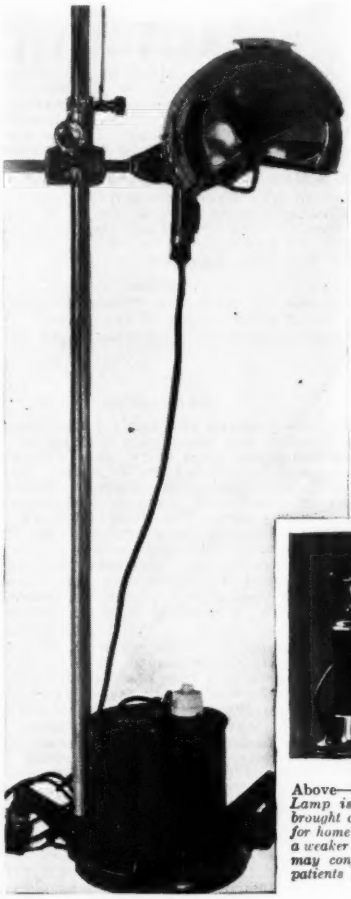

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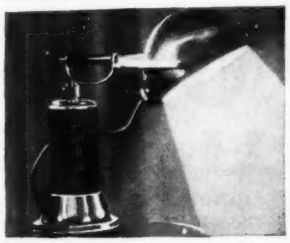
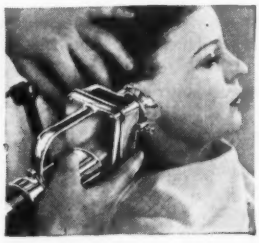
For your information, the various types of Hanovia Lamps are briefly described on this page. Literature, more fully explaining our different models, will be gladly sent on request. Just use the coupon for convenience.



Above—*The Home Model Alpine Sun Lamp is a safe, convenient apparatus brought out to introduce a safe modality for home treatment. This model produces a weaker ray than the clinical lamps. You may confidently recommend it to your patients for producing tonic and prophylactic effects*



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Name.....

Address.....

(Continued from page 694)

Dr. Hugh Reid, Stanford, aged 68, died March 18 of acute yellow atrophy of the liver.

Dr. Ernest Diggs, Shelbyville, aged 47, died April 22 of a skull fracture received in an automobile accident.

Dr. Charles McClure, Cox's Creek, aged 55, died March 26.

Dr. J. Edward Williams, Morton's Gap, aged 65, died April 14 of pneumonia.

LOUISIANA

Dr. Amedee Granger, Head of the Department of Radiology, Graduate School of Medicine of Tulane University, New Orleans, has been awarded the gold Palmes Universitaires by the French government for outstanding work in radiology. France awarded the silver Palmes Universitaires to Dr. Granger in 1921. He was also awarded the gold medal of the Radiological Society of North America in 1926.

The Louisiana Dermatological Society has elected the following officers for 1929: Dr. M. T. Van Studdiford, New Orleans, President; Dr. R. A. Oriol, New Orleans, Vice-President; Dr. T. A. Maxwell, New Orleans, Secretary-Treasurer.

Deaths

Dr. James A. Mangham, Ringgold, aged 74, died March 30 of acute edema of the lungs.

Dr. Lawrence Daly, Opelousas, aged 66, died in March.

MARYLAND

At the one hundred and thirty-first annual meeting of the Medical and Chirurgical Faculty of Maryland, held in Baltimore April 23-24, the following officers were elected: Dr. Henry M. Fitzhugh, Westminster, President; Dr. Joseph W. Holland, Baltimore, Dr. William T. Hammond, Easton, and Dr. Frank B. Hines, Chestertown, Vice-Presidents; Dr. John T. King, Jr., Baltimore, Secretary, and Dr. Charles E. Brack, Baltimore, Treasurer.

Dr. Charles C. Hedges, Assistant Director of Johns Hopkins Hospital, Baltimore, has been appointed Director of the Babies' and Children's Hospital, a unit in the Columbia University Medical Center.

Dr. and Mrs. Harry G. Timbres, Baltimore, will sail in September for India, to work in Santiniketan under the poet, Rabindranath Tagore. Dr. Timbres will be in charge of a new hospital and will supervise the health of the students in the college and assist in health work in the surrounding community.

Dr. William Sydney Thayer, Baltimore, has been awarded the honorary degree of doctor of laws by McGill University, Montreal. Edinburgh University also has awarded this degree to Dr. Thayer.

Dr. William H. Welch, Professor of the History of Medicine, Johns Hopkins, Baltimore, was honored with a dinner celebrating his seventy-ninth birthday. Dr. Welch is in excellent health and busily engaged in his new work.

Dr. Joseph C. Clark, for thirty years Superintendent of the Springfield State Hospital, Sykesville, has been relieved of work because of illness. In appreciation of his long service he has been appointed Superintendent Emeritus, and arrangements have been made for him to continue living in his present home at the Hospital.

Dr. Edwin P. Bledsoe, Medical Officer in charge of the U. S. Veterans' Bureau Hospital at North Chicago, Illinois, has been appointed Superintendent of Springfield State Hospital, Sykesville.

Dr. Allen K. Krause, Associate Professor of Medicine and Director of the Kenneth Dows Tuberculosis Research Laboratories, Johns Hopkins, Baltimore, has been appointed Director of the Desert Sanatorium and Research Institute, Tucson, Arizona.

Dr. Ferdinand C. Lee, Baltimore, and Miss Jane Palfrey Homer, Riderwood, were married April 17.

(Continued on page 36)

WHEN A DIABETIC SAYS:
"What can I eat that tastes good?"

One of the problems in diabetes is to keep the patient diet-happy! And Knox Sparkling Gelatine can be of real service.

As you will note in the recipe below, Knox Gelatine combines perfectly with the foods prescribed for the diabetic diet. It makes dishes that are appetizing and *different* to the eye and the taste. It supplies the diet variety that satisfies the patient's appetite and it supplies the food-bulk that the patient's hunger craves.

People suffering from diabetes really enjoy gelatine dishes—and they *can* enjoy them if they have our diabetic recipes prepared by one of the country's recognized dietitians. Remember, Knox Gelatine is free from *sugar*.

KNOX *is the real* GELATINE

Contains No Sugar

WINTER SALAD (*Six Servings*)

	Grams	Prot.	Fat	Carb.	Cal.
2 teaspoons Knox Sparkling Gelatine	4.5	4	....	....	....
¼ cup cold water	....	....	....	....	....
½ cup hot water	....	....	....	....	....
½ teaspoon salt	....	....	....	....	....
⅓ cup vinegar	....	....	....	....	....
1½ cups grated cheese	150	43	54	....	....
½ cup chopped stuffed olives	70	1	19	8	....
½ cup chopped celery	60	1	....	2	....
½ cup chopped green pepper	25	....	....	1	....
½ cup cream, whipped	75	2	30	2	....
Total		51	103	13	1183
One serving		8.5	17	2	197

Soak gelatine in cold water. Bring water and salt to boil and dissolve gelatine in it. Add vinegar and set aside to chill. When nearly set, beat until frothy, fold in cheese, olives, celery, pepper and whipped cream. Turn into molds and chill until firm. Unmold on lettuce leaf and serve.

SPINACH SALAD (*Six Servings*)

	Grams	Prot.	Fat	Carb.	Cal.
1½ tablespoons Knox Sparkling Gelatine	10	9	....	....	....
¼ cup cold water	....	....	....	....	....
1½ cups boiling water	....	....	....	....	....
2 tablespoons lemon juice	20	....	....	2	....
½ teaspoon salt	....	....	....	....	....
1½ cups cooked spinach, chopped	300	6	....	7	....
2 hard cooked eggs	100	13	10.5	....	....
Total		28	10.5	9	242.5
One serving		5	2	1.5	40

Soak gelatine in cold water and dissolve in boiling water. Add lemon juice, salt, strain and chill. When nearly set, stir in chopped spinach, mold and chill until firm. Serve on lettuce hearts or tender chicory leaves and garnish with hard cooked egg, cut lengthwise in sixths and sprinkled with paprika. Serve with mayonnaise.

If you agree that recipes like the ones on this page will be helpful in your diabetic practice, write for our complete Diabetic Recipe Book—it contains dozens of valuable recommendations. We shall be glad to mail you as many copies as you desire. Knox Gelatine Laboratories, 408 Knox Ave., Johnstown, N. Y.

Name..... *Address*..... *City*..... *State*.....

(Continued from page 34)

Deaths

Dr. Samuel Searle Houlton, Baltimore, aged 56, died March 24 of heart disease.

Dr. Edgar B. Britton, Baltimore, aged 76, died March 27 of myocarditis and emphysema.

Dr. Daniel Smith Lamb, Poplar Hill, died April 22 of pneumonia.

Dr. David Russell Talbott, Dunkirk, aged 53, died April 28.

Dr. Joseph Bruff Seth, St. Michaels, aged 51, died April 18 of cerebral hemorrhage.

MISSISSIPPI

Dr. Hugh A. Gamble, Greenville, has been elected President of the Mississippi State Medical Association.

Dr. Temple Ainsworth, formerly House Physician, Laurel General Hospital, Laurel, is now one of the Resident Physicians at the South Mississippi Charity Hospital.

Vicksburg was selected as the meeting place for the Mississippi State Medical Association in 1930.

Dr. May F. Jones, Sanatorium, who has been ill for some time from an attack of influenza, has gone to West Point, Virginia, to recuperate.

The State Children's Health Camp opened on June 3 with twenty-five children. The camp will operate for three months, caring for three different groups of children.

Deaths

Dr. Ernest Leonard Posey, Jackson, aged 40, died April 4 of chronic pleurisy and tuberculosis.

MISSOURI

The Dent County Medical Society has elected the following officers for 1929: Dr. J. C. Welch, Salem, President; Dr. Lloyd H. Hunt, Salem, Vice-President; Dr. W. E. Rudd, Salem, Secretary-Treasurer; Dr. J. C. Welch, Salem, Delegate, and Dr. A. T. McMurty, Salem, Alternate.

Dr. Kerwin W. Kinard, Kansas City, was elected President-Elect of the American Association for the Study of Goiter at its annual meeting in March. Dr. Kinard served as Secretary of the Association for four years.

Dr. R. B. H. Gradwohl, St. Louis, was ordered to the Navy Medical School, Washington, D. C., April 13, to give a course of instruction on the Schilling blood work. Dr. Gradwohl is a Lieutenant Commander in the Naval Reserve.

The St. Louis Medical Society is forming an orchestra exclusively for members of the Society. Dr. R. B. H. Gradwohl is the Chairman, and a well known conductor, Mr. Leonid Lenoardi, has volunteered to train and lead the orchestra.

Dr. Augustus G. Pohlman, Professor of Anatomy, St. Louis University School of Medicine, has been granted a year's leave of absence to engage in research on diseases of the ear in Buffalo in a laboratory equipped with instruments of precision on sound physics. Dr. Pohlman has studied sound physics for years, hoping to find a method of alleviating middle ear deafness.

Dr. Bransford Lewis, St. Louis, recently won an exhibition chess game from Mr. J. A. Anderson, the champion of St. Louis.

Deaths

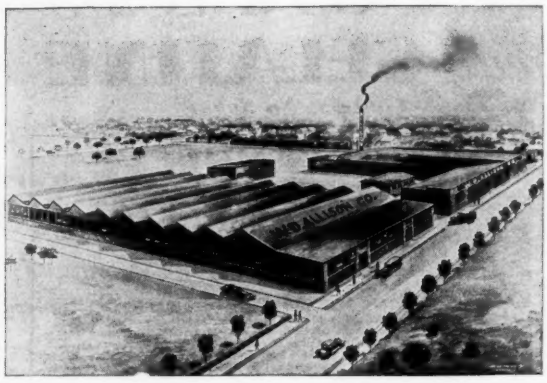
Dr. Harry Lamartine Fichtenkam, St. Louis, aged 81, died April 13 of intestinal obstruction and chronic myocarditis.

Dr. John A. Mitchell, Branson, aged 78, died March 25 of pneumonia.

Dr. Charles A. Todd, St. Louis, aged 80, died February 23.

(Continued on page 38)

OUR NEW HOME



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In *Neosalvarsan (brand of Neoarsphenamine) — the dependable original of Ehrlich— you are assured of maximum activity, that potency on which depends the value of the antiluetic agent.

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(Continued from page 36)

Dr. Jack Butler Grubbs, Farmington, aged 25, died April 18.

Dr. John C. Faris, Caruthersville, aged 47, died April 1 of pneumonia.

Dr. Marion L. Currier, St. Charles, aged 78, died March 23 of heart disease.

Dr. James J. Smcot, Bethel, aged 82, died April 21.

NORTH CAROLINA

At the annual meeting of the Medical Society of the State of North Carolina, held in Greensboro in April, the following officers were elected: Dr. L. A. Crowell, Lincolnton, President; Dr. William B. Murphy, Snow Hill, Dr. William E. Warren, Williamston, and Dr. Noah B. Adams, Murphy, Vice-Presidents; Dr. L. B. McBrayer, Southern Pines, Secretary-Treasurer (reelected).

Dr. H. C. Dodge, Chief of the Regional Offices of the U. S. Veterans Bureau, Washington, has been appointed Medical Officer in charge of U. S. Veterans Hospital No. 60 at Oteen.

The Biltmore Hospital, Biltmore, is building an additional wing to cost $125,000, to be known as the Battle Wing, honoring the late Dr. S. Westray Battle, first medical director of the institution. The Duke Foundation will contribute $50,000 toward the erection of this building.

It is reported that the Duke Foundation and the Rosenwald Foundation will build a dozen district hospitals for negroes in North Carolina.

The Tar Heel Sanitarium, Inc., has recently been organized and will be located about five miles from Greensboro. Dr. W. J. Meadows, Greensboro, will be in charge. The building will cost between $50,000 and $100,000.

Dr. L. B. McBrayer, Southern Pines, has been elected President of the Chamber of Commerce of that city.

Dr. K. P. B. Bonner, Morehead City, has been elected Mayor of that city.

Dr. P. P. McCain, Superintendent of the State Sanatorium, Sanatorium, was awarded the medal for 1928 given by the Moore County Medical Society each year for the best paper presented before that Society.

Dr. Alan R. Anderson, a son of Dr. Thomas E. Anderson, Statesville, has been made Dean of the New York Post-Graduate Medical School.

Deaths

Dr. J. G. Reynolds, Marion, died May 15 of heart trouble.

Dr. W. T. H. Brantley, Bethel, died May 15 of acute nephritis.

Dr. Edward P. Snipes, Jonesboro, aged 76, died April 30 of cerebral hemorrhage.

Dr. John Sweaney, Durham, aged 71, died March 23 of myocarditis.

Dr. William T. Shipp, Valdese, aged 65, died April 17 of cerebral hemorrhage.

OKLAHOMA

Dr. Lawson Hughes, formerly of Tonkawa, announces his removal to Tulsa.

Dr. Thomas McElvoy, Ponca City, has returned from a three months trip to Vienna and other European cities.

Dr. Allen C. Cramer, Ponca City, has been appointed Health Officer for Kay County.

Dr. G. M. Rushing, Durant, has been appointed Health Officer for Bryan County.

The New Border Hospital, Mangum, is nearing completion. This addition doubles the capacity of the Hospital.

The Noble County Medical Society has elected the

(Continued on page 40)

McKesson Universal Unit No. 100

Another
McKesson Advantage

An advantage of the intermittent principle of the McKesson apparatus is its ease of operation. If the patient breathes rapidly for a few moments, the gas in maintaining its pressure, speeds up delivery with the respirations; when respirations become slower, the apparatus slows up the delivery automatically—relieving the operator of constantly shifting the flow rate with every change or taking the consequences of either light anesthesia at one time or excessive waste of gas at the other.

Write for Complete Information

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Technical Appliance Co.

2226-36 Ashland Ave. TOLEDO, OHIO

Prevent Summer Diarrhea!

Free from pathogenic bacteria, the use of Dryco avoids milk-borne diarrhea, enteritis and other similar disorders. This is the season when such infections become increasingly prevalent. Prescribing Dryco now means the elimination of many summer feeding troubles. . . . . .

In cases of disturbed infantile metabolism, when various formulae fail to produce the desired result and nothing else agrees, try Dryco. It is well borne when other forms of milk are not tolerated and has proved its merit in thousands of difficult feeding cases over a period of many years.

Send for suggested feeding tables, Dryco samples and clinical data!

DRYCO

For convenience, pin this to your letterhead or Rx blank and mail.

THE DRY MILK COMPANY 15 PARK ROW, NEW YORK

BACILLUS ACIDOPHILUS CULTURE
(B. A. CULTURE)

For many years our Cultures have been largely used in "baby cases" throughout the South during the summer months.

B. B. CULTURE and
B. A. CULTURE (4-oz.) are at your service at the better prescription pharmacies.

B. B. Culture Laboratory, Inc.
Yonkers, New York

(Continued from page 38)

following officers for this year: Dr. T. F. Renfrow, Billings, President; Dr. J. W. Francis, Perry, Vice-President; Dr. B. A. Owen, Perry, Secretary-Treasurer.

The Okmulgee-Okfuskee County Medical Societies held the annual all-day clinic April 15 at Okmulgee.

Dr. Clyde W. Beson, Oklahoma City, has been appointed State Health Officer. He has been city physician for four years at Oklahoma City.

Dr. John P. Sudderth, Nowata, has been appointed Health Officer for Nowata County.

Deaths

Dr. W. H. Price, Eldorado, aged 50, died April 27.

Dr. Daniel F. Janeway, Stillwater, aged 77, died April 21 after a lingering illness.

Dr. James E. Webb, Tulsa, aged 72, died April 12.

Dr. John H. Beucler, Oklahoma City, aged 84, died March 17 of nephritis.

Dr. Charles Pickens Linn, Tulsa, aged 68, died March 25 of arteriosclerosis.

Dr. Andrew Jackson Smith, Pawhuska, aged 73, died April 5 of pneumonia.

SOUTH CAROLINA

At their annual meeting in May the South Carolina Medical Association elected the following officers: Dr. C. R. May, Sumter, President; Dr. G. P. Neel, Greenwood, First Vice-President; Dr. J. B. Johnson, St. George, Second Vice-President; Dr. J. F. Davis, Clinton, Third Vice-President; Dr. E. A. Hines, Seneca, Secretary-Treasurer (reelected). Councilors were elected as follows: First District, Dr. J. H. Cannon, Charleston; Third District, Dr. T. L. W. Bailey, Clinton; Fifth District, Dr. J. R. DesPortes, Fort Mill; Sixth District, Dr. M. R. Mobley, Florence; Seventh

(Continued on page 42)

Alkali Deficiency

Many diseases are complicated by an "acidosis." An important part in their treatment consists in replacing those elements needed to maintain the alkali reserve.

In clinical practice a rational and agreeable method of alkalinization is afforded in **Kalak Water.**

1 Liter of Kalak Water requires 710 cc. of a standard tenth-normal hydrochloric acid solution for neutralization of the bases present as bicarbonates or carbonates.

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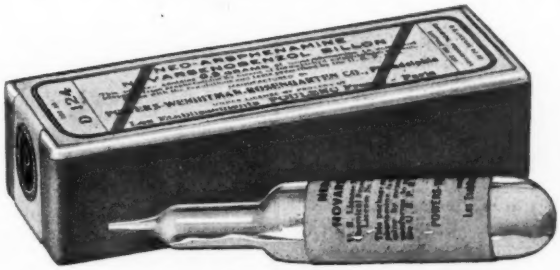

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Cystitis

℞ 5 to 15cc, by injection direct into the bladder.

C. R. BARD, Inc.

Exclusive Agents in the United States

79 Madison Ave. New York, N. Y.

(Continued from page 40)

District, Dr. T. R. Littlejohn, Sumter. The annual meeting next year will be held in Florence.

Deaths

Dr. Carroll Sumner Howell, Marion, aged 46, died February 27.

Dr. William Parks Barratt, Greenwood, aged 67, died April 9.

Dr. William Jacob Murray, Columbia, aged 73, died March 30 of pneumonia.

TENNESSEE

Dr. C. Sidney Burwell, Professor of Medicine, Vanderbilt University School of Medicine, Nashville, addressed the Staff Meeting of the Massachusetts General Hospital, Boston, March 14. The subject of his paper was "The Mechanisms of Heart Failure."

Dr. S. J. Platt, graduate of University of Tennessee Medical Department, has opened offices in Knoxville and will do general practice.

Dr. L. A. Absher, formerly of Portland, has removed to Knoxville.

Dr. W. S. Dooley has removed from Wilder to Crossville.

Dr. George Grant Henson, formerly of Miami, Florida, has located in Knoxville and will do general practice.

Dr. J. L. Raulston has removed from Richard City to Knoxville.

At the ninety-sixth annual meeting of the Tennessee State Medical Association held in Jackson on April 11 the following officers were elected: Dr. Leon T. Stem, Chattanooga, President; Dr. John A. McCulloch, Maryville, Vice-President for East Tennessee; Dr. Maurice L. Hughes, Clarksville, Vice-President for Middle Tennessee; Dr. James A. Price, Oakville,

(Continued on page 44)

All Through The Summer Months

Calcreose is the "stand-by" of many physicians when they require an intestinal antiseptic embodying the effectiveness of creosote.

Every physician reader of this Journal who is not familiar with the value of Calcreose in the treatment of enteritis and other intestinal disturbances during the summer months is invited to clip out and return the bottle opposite for liberal, complimentary test.

The MALTBIE Chemical Company, Newark, N. J.

MALTBIE *Calcreose*

Relieving the Busy Physician of Exacting Detail in Infant Feeding

RESULTS

SIMPLICITY

S. M. A. was designed to Simplify the physician's work of infant feeding and to make it more Effective.

May we send you samples?

S. M. A. was developed at the Babies and Childrens Hospital of Cleveland and is produced by its permission exclusively by The Laboratory Products Company.

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64 Gerrard St., East, Toronto

LPC

SAFE, SIMPLE INFANT FEEDING

HORLICK'S Malted Milk is safe and simple in infant feeding. Its successful use for nearly half a century has demonstrated the following outstanding advantages:

1 The readily assimilable state of its minerals promotes sound bone and tooth structure.

2 The light, flaky curds produced because of the modified nature of its milk constituent aid digestion.

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4 The exclusive Horlick process conserves the vitamin content of milk and malted grains unimpaired.

For samples address—HORLICK, Racine, Wis.

THE ORIGINAL MALTED MILK
HORLICK'S

(Continued from page 42)

Vice-President for West Tennessee; Dr. John Owsley Manier, Nashville, Treasurer (reelected); Dr. Harrison H. Shoulders, Nashville, Secretary (reelected); Dr. Hiram B. Everett, Memphis, Dr. Harrison H. Shoulders, Nashville, and Dr. Leon L. Sheddan, Knoxville, Delegates to the American Medical Asscciation. The next annual meeting will be at Nashville the secone week of April, 1930.

The Baptist Memorial Hospital Staff, Memphis, has elected the following officers: Chief of Staff, Dr. J. J. Shea; Vice-President, Dr. J. W. Bodley; and Secretary, Dr. M. W. Searight.

At a recent staff meeting the St. Elizabeth Hospital, Elizabethton, elected the following physicians to the staff: Dr. J. L. Cotrell, Dr. W. G. Frost, Dr. W. H. Longmire, and Dr. E. T. Pearson.

Dr. E. B. Clark has located at Sparta, after completing three months of special work at the Protestant Hospital, Nashville.

Dr. E. A. Adams, formerly of Danville, Pa., has taken charge of one of the surgical divisions of the Sanders-Warr Polyclinic at Memphis.

The new Medical Arts Building at Chattanooga was opened April 1. It is ten stories and modern in every respect. It is owned and occupied by the local medical men.

Construction will soon begin on the proposed ten-story Medical Building in Knoxville.

Cumberland-Putnam-Overton-White Counties Medical Society has elected the following officers for 1929: Dr. W. A. Howard, Cookeville, President; Dr. E. L. Mooneyham, Rock Island, Vice-President; Dr. H. H. Taylor, Cookeville, Secretary-Treasurer.

Franklin County Medical Society has elected the following officers for 1929: Dr. L. A. Templeton, Winchester, President; Dr. John P. Grisard, Winchester, Secretary.

Deaths

Dr. John Alexander Witherspoon, Nashville, aged

(Continued on page 46)

CAPROKOL

Affords Quick Relief in Urinary Infections

It does not irritate the bladder mucosa even when given in maximum doses and is equally effective in acid or alkaline urine.

Indicated in the treatment of—

Pyelitis—acute or chronic

Cystitis—acute or chronic

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Secondary Infections—associated with prostatitis or distant foci of infection.

Valuable as a prophylactic before and after instrumentation and operation.

Capsules for adults *Solution for children*

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BALTIMORE

New York Chicago New Orleans St. Louis Atlanta Boston
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Additional Uses for Protein Milk

THE use of Protein Milk in infant feeding is by no means limited to the treatment of the common fermentative Diarrhea—so called "Summer Diarrhea."

A wealth of clinical evidence gathered during recent years shows Protein Milk to be the food of choice for the premature infant, and for those types of nutritional disturbance characterized by hydrolability and inability to digest or assimilate normal formulae.

Protein Milk is most easily prepared and conveniently used if you recommend Merrell-Soule Powdered Protein Milk.

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Long special laced back Extension of soft material low on hips.

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Price $2.00

I. L. LYONS & CO., LIMITED

NEW ORLEANS, LA.

(Continued from page 44)

64, died April 28 after an illness of about two months.

Dr. William Butler Leē, Nashville, aged 72, died April 9 of angina pectoris.

Dr. Robert L. Hayes, Nashville, aged 59, died March 11 of septicemia.

Dr. James Talleyrand McColgan, Nashville, aged 83, died April 15 of nephritis.

Dr. William Bailey, Nashville, aged 65, died April 20 of paralysis agitans.

Dr. Marcus C. Wright, Knoxville, aged 55, died March 30 of injuries received in an automobile accident.

Dr. John Martin Arnold, Jackson, aged 77, died April 3 of cerebral hemorrhage.

Dr. Edward K. Blair, Fayetteville, aged 62, died March 25.

Dr. James Morton McLaughlin, Woodbine, aged 68, died April 10 of injuries received when struck by an automobile.

Dr. James T. Dyer, Mohawk, aged 70, died March 20.

Dr. John D. Shannon, Greenfield, aged 65, was killed April 25 when the automobile which he was driving was struck by a train.

TEXAS

Dr. J. B. Rawlings, El Paso, has been elected President of the Texas Public Health Association.

The Latham Hospital, Caldwell, had its formal opening on March 18. It is owned and operated by

(Continued on page 48)

Patient Types . . .

The Man on His Feet

Irregular and uncertain times for defecation may lead to hemorrhoids and more often to constipation. Cathartics aggravate the condition.

Petrolagar is very helpful in managing these cases. It brings about normal peristalsis in a natural way. It prevents the congestion of the hemorrhoidal veins caused by straining at stool.

Petrolagar is a mechanical emulsion of liquid petrolatum (65% by volume) and agar-agar, deliciously flavored and pleasant to take. It has many advantages over plain mineral oil. It mixes easily with bowel content, supplying unabsorbable moisture with less tendency to leakage. It does not interfere with digestion.

Petrolagar restores normal peristalsis without irritation, producing a soft-formed, normal stool consistency and real comfort to bowel movement.

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Gentlemen:—Send me copy of "HABIT TIME" (of bowel movement) and specimens of Petrolagar.

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MANHATTAN EYE SALVE COMPANY

Louisville, Kentucky

(Continued from page 46)

Dr. W. W. Latham and is the first institution of its kind in Burleson County.

The Oak Cliff Medical-Dental Building, Dallas, held its formal opening April 3.

The Waco Medical Arts Building, Waco, was opened April 4. This structure cost $450,000 and is modern in every respect.

The Dallas Southern Clinic Society was recently organized in Dallas by a group of physicians and officers elected were: Dr. O. M. Marchman, President; Dr. T. C. Gilbert, Vice-President; Dr. Curtice Rosser, Secretary, and Dr. G. E. Brereton, Treasurer. Dr. D. L. Bettison, Chairman, and Dr. John G. Young and Dr. Thomas J. Calhoun were appointed members of a committee to work out a constitution and by-laws for the new organization. It is the plan of the Society to hold one, two and three-day clinics, extending invitations to physicians of the Southern States.

The contract has been let for a $60,000 hospital at Sweetwater, and it is expected that it will be finished in eight months. It will be a four-story structure, arranged so that additional wings may be added as needed. The first unit will have thirty beds.

The Texas Interurban Club, an association of internists, held its annual session at Waco March 29. Dr. K. H. Beall, Fort Worth, is President of the Club, and Dr. C. T. Stone, Galveston, is Secretary-Treasurer. Membership in the club is limited to thirty, and twenty-six attended this meeting.

Dr. Ralph Robert Nowlin, San Antonio, was married to Miss Phronsye Lorraine Lynch, also of San Antonio, on March 9.

Dr. Henry M. Winans, Dallas, has been appointed Professor and Chairman of the Department of Medicine at Baylor University College of Medicine.

Deaths

Dr. Murff Franklin Bledsoe, Port Arthur, aged 49, died March 16.

(Continued on page 50)

ORGANOTHERAPY

can be effective only through the use of dependable endocrine products. The reputation and integrity of the manufacturer is the physician's only guarantee of reliability of those organotherapeutic products for which there is no chemical or biological assay. Every manufacturing process of all our products is supervised by our Analytical and Research Department.

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Marshaling scientific and medical opinion for the improvement of diet and of health

MODERN business intelligence realizes that the interests of an industry cannot be permanently advanced unless the public is benefited by such an advance. The Sugar Institute—representing an association of the cane sugar refiners of the United States—is proceeding on the belief that the position of the sugar industry cannot be permanently enhanced unless such enhancement results concurrently in improved diet and health for all ages and classes.

The Sugar Institute, under the guidance of eminent scientific authorities, is seeking to show the public in simple, understandable language, in more than 500 newspapers, how sugar as a flavor may be used to encourage the ingestion of many healthful foods so likely to be neglected in the inadequate diet.

Every effort is being made to discourage the public from gorging or overeating sugars or other sugar-containing foods.

The public is being advised not to eliminate sugar or any other food from the diet unless upon the advice of a physician.

The dangers of extreme dieting for unnatural weight reduction as pointed out by numerous physicians are being emphasized.

A constant drive for a varied, balanced diet is being carried on with special emphasis upon milk, fruits, vegetables and cereals.

Recipes, prepared by cooking experts of national standing, are being published to show how small amounts of sugar as nature's supreme flavorer relieve the natural blandness of many foods and make these healthful foods more acceptable and delightful to the taste of growing children and adults.

The Sugar Institute asks the cooperation of all physicians and health authorities who are sympathetic with its platform to help make it effective. Good food promotes good health. The Sugar Institute, 129 Front Street, New York, N. Y.

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INTEREST in small established modern tuberculosis sanatorium in good Southwest health resort can be bought by experienced tuberculosis physician. Owner's health demands giving up active charge. Address C. L. T., Southern Medical Journal.

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(Continued from page 48)

Dr. Bryant M. Harrison, Appleby, aged 64, died suddenly March 8 of acute dilation of the heart.

Dr. Wylie Cheney Cunningham, Dexter, aged 64, died March 9.

Dr. W. Walter Bouldin, Bay City, aged 62, died March 14 of cerebral hemorrhage.

Dr. G. Daniel Strickland, Cleburne, aged 72, died March 14.

Dr. William Washington Halbert, Corsicana, aged 48, died suddenly March 12.

Dr. Levingston Lindsay Shropshire, San Antonio, aged 72, died April 18 of carcinoma of the stomach.

Dr. Richard E. Grigg, Buckholts, aged 79, died February 17.

Dr. Benjamin Franklin Terry, Rising Star, aged 69, died April 4.

Dr. Walter W. Fowler, Ballinger, aged 70, died in March.

VIRGINIA

The South Piedmont Medical Society held its semi-annual meeting on April 16 and elected the following officers for 1929: Dr. I. Keith Briggs, South Boston, President; Dr. Harry Pritchett, Danville; Dr. John W. Carroll, Lynchburg; Dr. C. W. Tucker, Drakes Branch; Dr. H. H. Hurt, South Boston, Vice-Presidents; Dr. George A. Stover, South Boston, Secretary-Treasurer (reelected).

Dr. Leta J. White, Assistant Medical Inspector for the Richmond City Health Department, Richmond, has resigned her position and will go to the Children's Hospital, Philadelphia, for a year's study.

Dr. P. K. Graybill, who has spent the past several years in Arizona, will return to Virginia for the summer, and will have headquarters at Fincastle.

Dr. Horace Taylor Hawkins, Waynesboro, was married to Miss Avis Curtis Bernard, Richmond, April 22.

Dr. E. Claude Jamison, Rocky Mount, was married to Miss Ella Virginia Marshall, Mica, April 27.

Dr. Richmond J. Beck and Dr. Regena C. Beck, Richmond, have opened offices at 1103 West Franklin Street. Dr. Richmond Beck will limit his practice to neurology and psychiatry.

Dr. W. J. Rollins, Richmond, who for the past two years has been Resident Surgeon at University Hospital, has removed to Macon, Georgia, and will limit his practice to surgery.

Dr. W. W. Insley, formerly of Christiansburg, has removed to Roanoke.

Dr. W. W. Seward, Surry, was recently elected President of the Bank of Dendron, Virginia.

At the annual meeting of the Richmond School Board, Dr. Roshier W. Miller was elected Chairman and Dr. Clifton M. Miller, Vice-Chairman.

(Continued on page 52)

HEXALET "RIEDEL"

The Better Acid Medium Urinary Antiseptic

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Allays severe burning and has a soothing effect in kidney and bladder condiitons without causing hematuria when taken for a long period of time. To clear shreds and pus in chronic and non-specific cases.

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Sometimes quicker results can be obtained in obstinate cases by combining Lacto Dextrin with Psylla seeds (plantago psyllium). The latter provides bulk and lubrication and so combats constipation and hastens the passage of Lacto Dextrin into the colon.

The interesting story of the use of Lacto Dextrin alone or in combination with Psylla is told in the book, "A Practical Method of Changing the Intestinal Flora." We shall be glad to send you a copy together with clinical trial packages on request.

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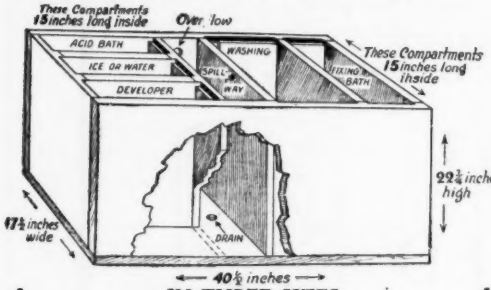

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Name.....

Address.....

(Continued from page 50)

Dr. B. R. Caldwell, of the U. S. Veterans Bureau, has moved his offices from Washington, D. C., to Alexandria. He will continue his connection with the Bureau as specialist in tuberculosis on the Advisory Group, and will also do consultation work.

Dr. James W. Keever, recently Resident Physician at Pine Camp Hospital, Richmond, has removed to Hickory, N. C., and will do general practice.

Dr. Douglas Vanderhoof, Richmond, has been appointed as a member of the Board of Visitors of the Medical College of Virginia.

Dr. Caleb S. Stone, Jr., St. Louis, has been appointed Resident Surgeon at the University Hospital, Richmond.

Deaths

Dr. Joseph M. Burke, Norfolk, aged 68, died April 5 of carcinoma of the pancreas.

Dr. E. L. Marshall, Lynchburg, aged 70, died May 22.

Dr. Benjamin L. Dillard, Scottsville, aged 71, died April 12 of heart disease.

WEST VIRGINIA

At the sixty-second annual meeting of the West Virginia State Medical Association held at Martinsburg May 23, the following officers were elected for this year: Dr. Walter E. Vest, Huntington, President; Dr. A. B. Eagle, Martinsburg, First Vice-President; Dr. R. V. Shanklin, Gary, Second Vice-President; Dr. F. T. Scanlon, Morgantown, Third Vice-President; Dr. T. M. Barber, Charleston, Treasurer; Dr. C. G. Morgan, Moundsville, Dr. C. H. Hall, Elkins, Dr. S. S. Hall, Bickhannon, Dr. O. B. Biern, Huntington, Dr. H. G. Steele, Bluefield, and Dr. H. A. Walkup, Mount Hope, Councillors. White Sulphur Springs was selected as the meeting place for 1930.

Dr. J. G. Pettit, Hopemont, was elected President of the West Virginia Tuberculosis Association for 1929.

THE STANDARD
LOESER'S INTRAVENOUS SOLUTIONS
CERTIFIED

LOESER'S INTRAVENOUS SOLUTION of IRON and ARSENIC

A standardized sterile solution, 5 cc. contain 64 mg. (1 grain) of Colloidal Iron Cacodylate. In hermetically sealed Jena glass ampoules.

In 12 years, steadily increasing numbers of physicians have demonstrated the safety and practicability of its intravenous injection and therapeutic value in the treatment of all Secondary Anaemias following Malaria, Pellagra, Influenza and other infections as well as Surgical Procedure.

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THERE is a unanimity of opinion among physicians upon the use of Mead's Dextri-Maltose.

It is shared alike by the pediatrist whose practice is confined exclusively to the feeding of infants, and the general practitioner whose work covers a multitude of different cases.

An abundance of clinical evidence has proved it is well tolerated, easily assimilated and generally productive of satisfactory results. In combination with various cow's milk and water modifications it has probably *successfully* been used to feed more infants than any other carbohydrate offered the medical profession.

A few infants can tolerate *any* carbohydrate. But *any* carbohydrate can not be fed all babies under all conditions. The success characterizing the use of Mead's Dextri-Maltose in the combined experience of infant feeders is that it will successfully feed *more* infants than any other now in use.

Sample and Literature on Request.

MEAD JOHNSON & CO.
Evansville, Indiana

MEAD'S

THE MEAD POLICY

MEAD'S infant diet materials are advertised only to physicians. No feeding directions accompany trade packages. Information in regard to feeding is supplied to the mother by written instructions from her doctor, who changes the feedings from time to time to meet the nutritional requirements of the growing infant. Literature furnished only to physicians.

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announce that they will shortly be ready to supply

VIOSTEROL, P. D. & CO.

(Irradiated Ergosterol)

(Licensed under the Steenbock patent administered by the Alumni Research Foundation of the University of Wisconsin)

Viosterol, P. D. & Co., will be released for sale to the drug trade on July 25, 1929. Your druggist may not have it in stock on that date, but he can get it for you on short notice.

Viosterol, P. D. & Co., will be supplied in the form of a vegetable oil solution of irradiated ergosterol standardized to an antirachitic (vitamin D) potency of one hundred times that of high-grade cod-liver oil. It will be furnished in 5-cc. and 50-cc. packages.

Viosterol is the name adopted by the Council on Pharmacy and Chemistry of the American Medical Association to designate preparations of irradiated ergosterol.

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